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VALUE OF OXYGEN TREATMENT AFTER THYROIDECTOMY*

SAMUEL F. HAINES, M.D. AND WALTER M. BOOTHBY, M.D.

ROCHESTER, MINNESOTA

IN a previous paper on oxygen therapy we called attention to an hitherto unobserved phenomenon, a sharp, crisis-like drop in temperature which usually occurred following the administration of oxygen to patients whose temperature was elevated and who were cyanotic as the result of pulmonary edema, or bronchopneumonia. We also reviewed the literature on the physiological factors and the anatomical arrangement of the bronchi and alveoli which in these conditions are unfavorable for the complete oxygenation of hemoglobin. This drop in temperature, being an objective sign of the beneficial influence of properly administered oxygen in the appropriate case, has served to emphasize the harmful effect, as pointed out by Haldane, of allowing even a mild degree of anoxemia to persist. Furthermore, in cases of hyperthyroidism, anoxemia may develop easily and lead to more serious consequences because of the relatively increased consumption of oxygen. Asher and Duran caused hyperthyroidism in rats by the administration of thyroid extract and found an increased sensitiveness to oxygen deficiency. Similarly, Asher and Streuli found that rats whose thyroid gland had been removed did not show any of the phenomena of suffocation exhibited by normal animals in atmosphere deficient in oxygen.

Auer and Gates, and Leo Loeb, in their studies of the causes of pulmonary edema in rabbits following intratracheal administration of epinephrine, found that the edema could be prevented by any method which diminished the increased negative intrapulmonary pressure caused by the administration of the drug. Loeb emphasized that the more intense the inspiratory movements, the greater the tendency to withdrawal of fluid from the blood, a tendency which would be favored, as pointed out by Johnson, by overdistention of the pulmonary vessels from temporary incoordination of the volume output of the right and left ventricles (Welch).

Not enough attention has been paid to the theory of acute pulmonary edema advanced in 1878 by Welch, who was then working in Cohnheim's laboratory at Breslau. In 1904, Welch (in a personal communication to Meltzer) restated his theory of a mechanical explanation of acute pulmonary edema as follows: "A disproportion between the working power of the left ventricle and of the right ventricle of such character that, the resistance remaining the same, the left heart is unable to expel in a unit of time the same quantity of blood as the right heart."

Partial tracheal obstruction is occasionally noted following thyroidectomy,

* Read before the American Association for the Study of Goiter, Denver, Colorado, June 18–20, 1928.

usually as a result of excessive tracheal mucus, with or without laryngeal edema or spasm. Cardiac disturbances not infrequently follow thyroidectomy in cases of hyperthyroidism and whereas these disturbances are as a rule not apparently serious in themselves, they may be contributing factors to the production of pulmonary edema, particularly if there is interference to the free passage of air to such a degree as to prevent efficient aeration of the blood. The use of oxygen in such cases may, by decreasing the force of respiration and overcoming cyanosis, be a means of breaking a vicious circle at its most vulnerable point.

We have been particularly watchful of patients with hyperthyroidism both before and after operation in an effort to detect the slightest degree of cyanosis in order to institute early treatment in the oxygen chambers. We have also treated in the oxygen chamber patients with complications which if more extreme would have resulted in detectable cyanosis. In this group are included patients with pulmonary edema, early bronchopneumonia, and mechanical obstruction to the air passages by excessive mucus in the trachea, distortion of the trachea from pressure or vocal-cord paralysis. In many instances, cyanosis will not be apparent if the patient is lying quietly, but will become so on his sitting up in bed, with the attendant increase in oxygen consumption. Also the cyanosis may be intermittent; often it is present only during sleep, particularly in cases of disturbances of innervation of the vocal cords. In these cases the patient's conscious effort during waking hours will keep the larynx sufficiently open to prevent cyanosis, but anoxemia may develop during the relaxation of sleep. While the patient is awake he may be able to clear the trachea of mucus frequently enough to prevent much interference with respiration but while he is asleep it may accumulate sufficiently to cause considerable cyanosis, especially if at the same time there is a falling back of the tongue and lower jaw.

It has been our experience that patients with cyanosis or on the verge of cyanosis are much more comfortable following admission to the oxygen chamber. Undoubtedly in many cases analyses of blood gas would show slight oxygen desaturation in those patients who do not show clinical cyanosis. Besides the patient's increased comfort in the oxygen chamber there is often a characteristic crisis-like drop of from 2°F. to 6.5°F. in temperature in the first six to twelve hours. There is usually a drop in the respiration rate, and especially a diminution in the exertion of respiration with its attendant decrease in the intrapulmonary negative pressure, and therefore a lessened tendency to the development of pulmonary edema. The pulse also is usually slower.

The present study includes 91 cases of exophthalmic goiter, hyperfunctioning adenomatous goiter, adenomatous goiter without hyperthyroidism, and carcinoma of the thyroid, in which partial thyroidectomy had been performed and the patients, for various reasons, were sent to the oxygen chamber following operation. These cases represented a large proportion of the serious postoperative reactions from more than 5000 cases of goiter in which operation was performed during the period from January, 1926, when the oxygen chambers were first completed, to June, 1928. Almost all the patients with goiter who died during this time from any cause had been admitted to the oxygen chambers. Of these 59 had exophthalmic goiter, 27 hyperfunctioning adenomatous goiter, 4 adenomatous goiter without hyperthyroidism and 1 carcinoma of the thyroid gland. Of these patients 67 lived and 24 died. The oxygen concentration in the chamber varied in different cases. As a rule, oxygen was kept at a concentration of 50 to 60 per cent. If a patient's cyanosis persisted, higher concentrations were used, but this was rarely necessary, and probably concentrations of more than 60 per cent were not of any particular advantage to the patient. As the clinical condition

improved, the oxygen tension was gradually decreased to 30 per cent before the patient was removed from the chamber. Binger has shown that irritation of the lungs or other untoward effects are not produced by concentrations less than 70 per cent.

The most striking effect to be noted from the bedside charts of patients suffering from pulmonary edema, bronchopneumonia, slight cyanosis or elevation of temperature is the drop in temperature following admission to the oxygen chamber. Of the 91 cases there was a drop in temperature in the first twelve hours in the chamber in 75. The greatest drop in temperature in twelve hours was 6.5°, and the average drop was 2.1° during the first twelve hours. Of the patients who lived, the average drop was 2.4°, and of those who died, 1.5°. It is interesting to compare these figures with averages based on the pathological condition present. The most marked drop in temperature was either in cases of pulmonary edema or of bronchopneumonia; there were 47 of these and the average drop in twelve hours was 2.8°, as compared with 2.1° for the entire group. That this drop in temperature is not a chance or coincidental phenomenon is shown by the fact that in 37 of the 91 cases a drop of 3° or more occurred in the first twelve hours. Conversely, following the patient's removal from the chamber, there was often an immediate rise in temperature above the previous average level. This could be lowered again by readmission to the chamber. In 55 of the 67 patients who lived, there was such a rise within twelve hours of removal from the chamber; it did not, however, prove serious in any case in this series. The greatest rise was 4°, and the average of the 67 cases was 1.7°. In all cases there was on each succeeding day a gradual decrease so that after from two to four days the temperature was nearly or quite normal. Patients with labored respiration from frank pneumonia or obstruction of the upper respiratory tract from whatever cause were greatly relieved immediately by the oxygen treatment. In the latter group tracheotomy was frequently avoided; in the 23 cases in which obstruction was the important complication it was performed only four times. There were only 2 deaths in the 23 cases; in 2 frank bronchopneumonia was also present.

In 65 of the 91 cases definite cyanosis was present on the patient's admission to the chamber. The average drop in temperature was 2.6° in twelve hours. The degree of cyanosis was slight in most cases, especially in the more recent ones, as we have considered it the important indication for the use of oxygen, and have made an effort to institute treatment at its earliest manifestation. The cyanosis was controlled at once in all cases but in a few instances it reappeared even in concentrations of oxygen from 50 to 60 per cent as the pneumonia progressed in both lungs. In cases in which it was necessary to use progressively higher concentrations of oxygen to control the cyanosis and in which finally it was impossible to control it, all the patients died. At necropsy the pulmonary involvement was found to be far more extensive than is usually noted at necropsy. This fact, as emphasized by Robertson in his discussion of the necropsy reports, must not be misinterpreted to mean that the oxygen treatment aggravated the condition. The explanation lies in the fact that these patients were kept alive several days longer than they otherwise would have lived, and as the bronchopneumonia was of the progressive type sufficient time elapsed for this extensive involvement to develop.

In a few cases oxygen has been administered with the oxygen tent alone, but these cases have not been included in this series. We have used the tent only when the two chambers connected with our service were occupied. If patients are not restless, and do not need much nursing attention, the results of treatment in the tent may be perfectly satisfactory. Occasionally a patient who is treated with

difficulty in a tent will be easily cared for in the chamber. In such cases, of course, the advantages of the chamber over the tent can be more clearly observed and the drop in temperature and pulse is much greater after admission to the chamber than had occurred after admission to the tent. The use of the tent necessitates more constant technical attention and more frequent analysis of the oxygen, but if it is properly managed and the patient is cooperative, the results may be satisfactory. Wilder has reported on a large series of cases following abdominal operation in which the oxygen tent has been used with very gratifying results.

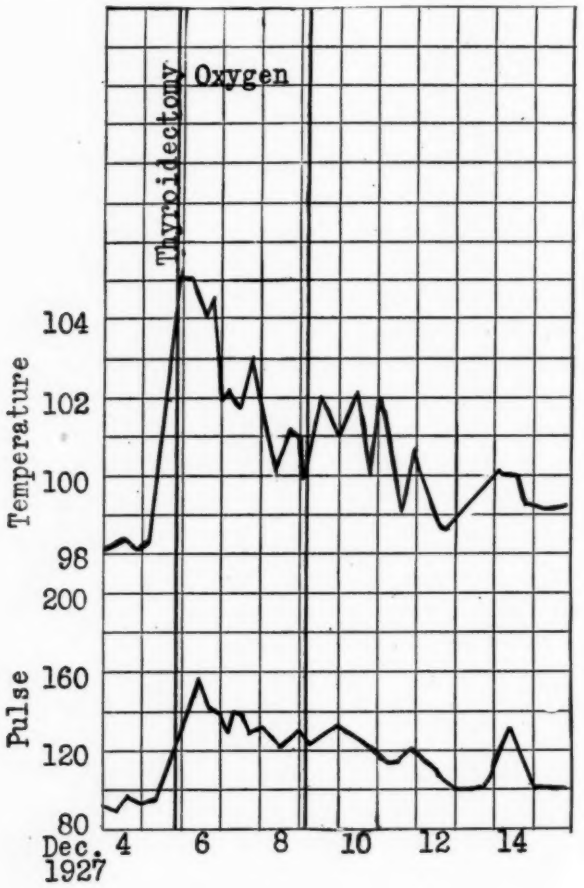

Fig. 1.

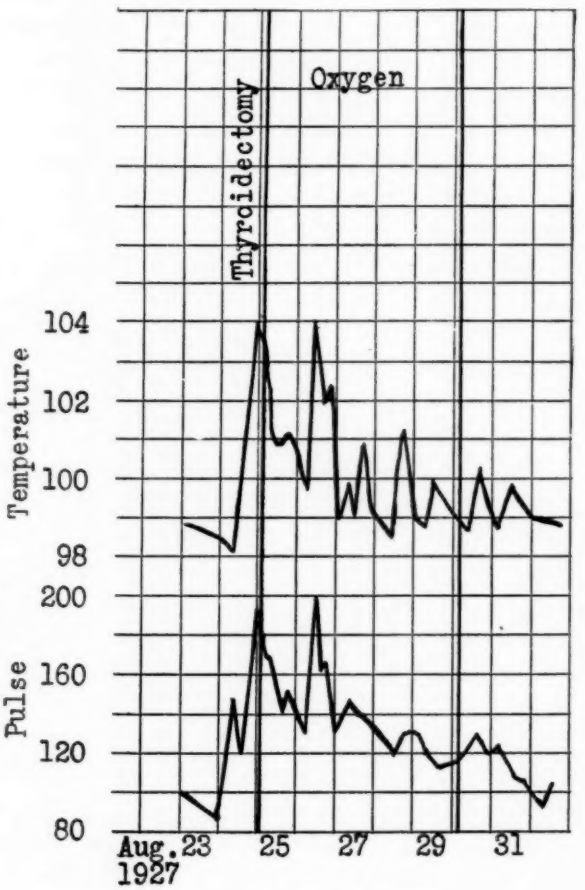

Fig. 2.

A direct comparison of mortality rates in the treated and untreated groups has not appeared feasible, since the surgical mortality rate in exophthalmic goiter (Pemberton) has been less than 1 per cent for several years. Various other factors influencing the mortality rate in previous years would also prevent a satisfactory comparison. The beneficial effect, therefore, of oxygen therapy, so far as this study is concerned, must be determined by clinical judgment and observation rather than by statistical methods. We do not feel hesitancy, however, in stating that the mortality from goiter during the last two years was somewhat lower than it would have been without the use of oxygen.

CASE REPORTS

Case 1. A woman, aged thirty-three, had had a moderately severe exophthalmic goiter for four years. At the time of examination the basal metabolic rate was +34 per cent. Preoperative treatment with compound solution of iodine was carried out over a period of twelve days, after which partial thyroidectomy was

performed. On the second day, the temperature had risen to 104.8°F.; the patient was quite ill, very uncomfortable, and slightly cyanotic. A few râles were heard at the left base of the chest, but evidence of consolidation was not present. The patient was admitted to the oxygen chamber. The cyanosis disappeared immediately and she was more comfortable. There was the usual drop in temperature and pulse, the temperature falling 3° in twenty-four hours, and reaching 100°F. after three days in the chamber. After removal from the oxygen chamber, there was the usual slight rise in temperature (Fig. 1), but serious reaction did not occur. This patient was considered much improved by oxygen treatment.

Case II. A woman, aged twenty-one, with a severe exophthalmic goiter of six month's duration and a basal metabolic rate of +59 per cent was treated with iodine prior to operation. During this time epidemic parotitis developed from which recovery was uneventful, although there was a severe reaction. After several days of normal temperature, partial thyroidectomy was performed. The day after operation the temperature was 104°F. When the patient was at rest, there was slight cyanosis, but this was much more marked after she moved about in bed. She was admitted to the oxygen chamber, where she was much more comfortable, but there was considerable rise in temperature daily (Fig. 2), and the reaction seemed to be of the infectious type. On the fourth day there were frank signs of pneumonia over the lower left lobe.

Case III. A woman, aged sixty-seven, had had exophthalmic goiter for two months, and a basal metabolic rate of +72 per cent. Because of the intoxication, she was kept under treatment with iodine for three weeks, after which partial thyroidectomy was performed. On the second day, the temperature rose to 103°F., and the pulse to 200. The auricles were fibrillating. Râles were heard at the right base, but consolidation was not evident. There was slight cyanosis. After admission to the oxygen chamber the patient was more comfortable, the pulse fell, cyanosis disappeared, and the temperature dropped 4° in twelve hours. During the first few days the sputum was blood-streaked but frank consolidation was not discovered. The course is shown in Figure 3.

Case IV. A girl, aged twenty, had suffered from an extremely severe exophthalmic goiter for more than three years. She was admitted

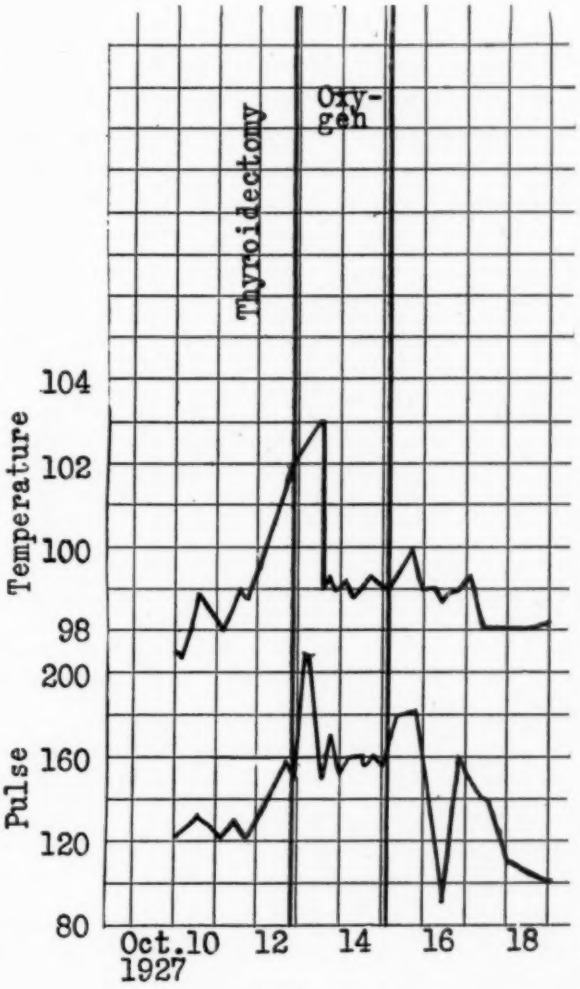

Fig. 3.

during the stage of a gastrointestinal crisis, at which time she was so weak that she could not sit up in bed. After prolonged medical treatment with iodine, partial thyroidectomy was performed. There was a severe hyperthyroid reaction with a sharp rise in pulse and temperature, and slight cyanosis of the fingernails. She was admitted to the oxygen chamber the day of operation. The usual drop in pulse and temperature occurred, but on the second day the temperature rose to 102°F., and there was distinct evidence of pneumonia in the left lower lobe. After a rather severe reaction, the temperature became normal on the ninth day, after which convalescence was uneventful. In this case, the patient was in an extremely poor condition due to long-continued severe

exophthalmic goiter. It is doubtful if she could have withstood the pneumonia without the benefit of oxygen.

SUMMARY

Oxygen was administered to patients having severe reactions after thyroidectomy, especially those with cyanosis resulting from pulmonary edema, bronchopneumonia, and laryngeal or tracheal obstruction. The patients showed, as a rule, marked subjective improvement. Usually the fever subsided rapidly. Pulse and respiration rates were lowered. Dyspnea was partially or completely relieved. It is probable that the development of pneumonia was prevented in many instances. The observations suggest that not infrequently death may be prevented by efficient oxygen treatment in suitable cases.

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THE USE OF IODINE IN HYPERTHYROIDISM*

ARNOLD S. JACKSON, M.D.

MADISON, WISCONSIN

IN the light of our present knowledge, of the use of iodine in hyperthyroidism, it seems to me that the following twelve questions offer material for discussion:

1. Is iodine *always* of value in exophthalmic goiter; that is, do certain patients with this disease fail to respond to iodine therapy?

2. Do all patients with hyperthyroidism improve with iodine? What effect has iodine in cases of toxic adenoma?

3. Does iodine initiate hyperthyroidism in non-toxic adenoma?

4. May iodine hyperthyroidism develop in any adenomatous goiter? Is the dosage of iodine or the time element important?

5. Is the amount of iodine used in the preoperative treatment of exophthalmic goiter an important factor?

6. When iodine is given during the preoperative preparation of patients with exophthalmic goiter what is the most favorable time for operation?

7. How may iodine be best administered during and following operation?

8. Does the use of iodine eliminate the necessity for ligation or stage operations in exophthalmic goiter?

9. What effects follow the prolonged use of iodine in hyperthyroidism?

10. Does the pathological picture of exophthalmic goiter ever show a state of complete reversion from hyperplasia to colloid following the use of iodine?

11. How long should iodine be continued postoperatively? Will prolonged use be beneficial or harmful?

12. What part does iodine play in hyperthyroidism on recurrence of symptoms?

I am presenting the problems briefly and trust that this will induce discussion which will emphasize certain factors that may be of extreme importance to those interested in the study of thyroid diseases.

As regards the value of iodine in exophthalmic goiter, it has been my experience that it has always proved effective in the primary treatment of this disease. There has been considerable discussion on this point, some physicians reporting that certain patients failed to respond to treatment. Although my experience has been limited to approximately 300 cases, there has been an almost immediate and remarkable response in every instance. In fact, so certain is improvement to occur that if the diagnosis is correct one can safely predict to the patient the progress of his condition. A patient may be brought in on a stretcher who has been bedridden for a month or longer while undergoing treatment for a supposedly bad heart. Is there any greater satisfaction than to be able to inform him that within a few days he will again be walking and on the way to recovery? I cannot help feeling that when iodine has not proved beneficial there has been an error in diagnosis, or in its use.

Do all patients with hyperthyroidism improve with iodine? My observation has been that they do not. This question again brings up the problem of classification, and the discussions which this subject called forth at the 1927 meeting of the American Association for the Study of Goiter will explain the futility of any attempt to renew it. I was surprised, recently, to read a report[1] from workers at the University of Michigan to the effect that iodine is satisfactory in the preoperative treatment

[1] YOUMANS, J. B. and KAMPMEIER, R. H. Effect of iodine in toxic adenoma. *Arch. Int. Med.*, 41:66, 1928.

* Read before the American Association for the Study of Goiter, Denver, June 18–20, 1928.

of almost every case of toxic adenoma. Certainly this has not been my experience. In fact, my records will show cases in which not only the basal metabolic rate has increased, but the symptoms of hyperthyroidism have become intensified through the use of iodine in toxic adenoma. In occasional instances some improvement has been apparent although the influence of rest and symptomatic treatment may have been a factor. Never in my experience has the brilliant response to iodine been apparent as has been observed in exophthalmic goiter. Naturally, when exophthalmic goiter is associated with adenomatous goiter, as is true in about 1 in 4 cases, toxic symptoms rapidly subside with iodine.

As regards iodine hyperthyroidism I can only reaffirm my view that hyperthyroidism may be initiated in previously non-toxic adenomatous goiter by the indiscriminate use of iodine. I have attempted to show that this condition is a distinct clinical entity and should not be confused with either exophthalmic goiter or toxic adenoma. The great amount of evidence that has accumulated in this country as well as abroad argues against the idea that this condition is one of theoretical rather than practical consideration. Numerous workers in this country have called attention to the increased incidence of this condition, and last year at the international goiter convention in Switzerland special attention was given the subject. Several hundred cases of iodine hyperthyroidism have now been reported in the literature, including 3 fatal cases which I have observed. This problem is one which impresses not only the laity but the profession as well with the fact that iodine is a drug and not a food, that it should not be used indiscriminately even in children, and, except in pregnancy or in preparation for thyroidectomy, it should not be used either in large amounts or for long periods of time by persons having adenomatous goiters.

It is true that certain persons having adenomatous goiter appear to have a definite resistance to the deleterious effects of iodine. Experiments have been made to prove that hyperthyroidism cannot be produced by iodine in adenomatous goiter. Certain persons will tolerate iodine apparently indefinitely with no harmful effect; in other cases that I have observed, even in children, Lugol's solution or patent medicines containing iodine have initiated toxic symptoms in less than two months. Small amounts of iodine may be tolerated for a considerable period of time in others, until a sudden accumulative effect seems to occur.

In 1922 Plummer made certain the wonderful results to be obtained with the use of iodine in conjunction with surgery in the treatment of exophthamlic goiter. At that time, I believe, the point was made that equally good results could be obtained with small as well as large doses of iodine. An early experience led me to believe that more immediate and marked improvement occurred with larger than with smaller amounts of iodine. Consequently the average dose given our patients has been 60 drops a day for a week. On the day of operation and on the day following this amount is at least doubled. Not only has the response to treatment seemed to be more speedy, but the degree of postoperative reaction has appeared to be considerably lessened. Likewise in the majority of cases the most favorable time for operation has seemed to be during the second week of preparation rather than later. However I realize that there is considerable room for discussion and I shall be interested to hear the views of others on this point. Naturally, patients who have been bedridden for some time, those who are extremely emaciated, or those in whom cardiac decompensation has occurred, may require a longer period of preparation.

In spite of the marked benefit from the use of iodine, certain patients with exophthalmic goiter continued to show a considerable degree of reaction following primary hyperthyroidism. Although no

fatal consequences resulted from this condition, the symptoms were often sufficient to cause considerable apprehension. Still, I always felt that if sufficient iodine could be retained the situation could be controlled. The most annoying factor was the repeated emesis and the occasional distressing tracheitis. Both of these conditions have been overcome by a method which I have been using during the past year and which I feel will be of general interest. In my experience no single factor with the exception of iodine itself has proved of such valuable aid in the treatment of this condition.

The plan which we have evolved is to introduce a duodenal tube on the evening preceding operation. This is done in all of the most toxic cases or those in which trouble is anticipated. This procedure permits the constant introduction of large amounts of fluid, iodine, and nourishment, not only during the morning of operation and the night preceding, but also during the period the patient is on the operating table, and the first afternoon which formerly was likely to be stormy. During the operation when the metabolic rate might be expected to be highest and the tendency to hyperthyroidism greatest, the patient is constantly receiving glucose, orange juice, and iodine or any other desired liquid. Under the influence of morphine and scopolamine the patient may quietly enjoy his breakfast the same as the other patients but without the trouble of swallowing. Moreover, the method is so simple as scarcely to require the services of a nurse, since the suspended container may be filled at the start with sufficient fluid to last during the operation. It has been our experience that even in the most toxic cases, following primary thyroidectomy, these patients experience little or no reaction after returning to their rooms. The temperature may rise to 101° F. and the pulse rate to 120, but seldom higher. Postoperative emesis and tracheitis are virtually eliminated. This method is also ideal in the treatment of cases with gastrointestinal crisis.

At this point I wish to reiterate the well-known and yet not generally understood fact that the prolonged use of iodine in exophthalmic goiter will lead to serious consequences. Among the several cases of this kind recently observed was one in which following a period of marked improvement, there developed cardiac decompensation and auricular fibrillation, another in which after a year's treatment the systolic blood pressure had increased to 268, and another in which hemiplegia had occurred. We should make it one of our duties to educate the profession and laity as well to the dangers of the prolonged use of iodine. While it may not be the responsible factor, still the disease progresses just as if no iodine had been used.

During the past year a valuable observation has been made, that those patients who appear to have developed a tolerance for iodine may again be restored to fair condition for operation by having the dosage of iodine greatly increased.

In a previous study of the pathological picture the fact was noted that iodine causes a reversion of hyperplasia to colloid, but that the change is never complete even after the prolonged use of iodine.

Since 1924 only one ligation operation has been performed at our clinic and in this instance no benefit was derived from it, nor was it expected, since the operation was done to determine the patient's ability to withstand thyroidectomy. Although it may seem a radical procedure, I believe that primary thyroidectomy may be safely performed in every case, provided there has been a proper period of preparation, the facilities are adequate, and the suggestions which I have just enumerated are carried out. It is true that we have lost patients under this plan, but I cannot attribute death to hyperthyroidism.

The time element in postoperative treatment with iodine is an involved question. In general the time is determined by the

condition of the patient at the time of operation, the amount of gland preserved, and the rapidity of recovery. The environment to which the patient returns is also a factor. If the metabolic rate has returned to normal within a month after operation, and the patient is gaining satisfactorily I see no reason for continuing the use of iodine. If only a small portion of the gland has been preserved (and no more should be saved than is absolutely necessary) I believe that long continued use of iodine may lead to the development of hypothyroidism, as has been suggested in the literature.

Although in my experience patients with primary hyperthyroidism are only temporarily benefited by the use of iodine, I feel that recurrence of hyperthyroidism following operation has been definitely and permanently prevented. Furthermore, the use of iodine over a few months' time does not seem to lead to serious consequences, although I believe prolonged use will. However, in most cases no benefit is derived and there should be a second operation.

I have briefly presented these points in the hope that they will stimulate discussion which will be helpful to all who are interested in further progress in the treatment of hyperthyroidism.[1]

[1] Jackson, A. S. Goiter and Other Diseases of the Thyroid Gland. N. Y., Paul B. Hoeber, Inc., 1926.

BORDERLINE HYPERTHYROIDISM*

HOWARD M. CLUTE, M.D., F.A.C.S.

BOSTON

IT is now quite generally accepted that if hyperthyroidism is present, the basal metabolic rate is elevated above normal, and, conversely, that if the basal metabolic rate is normal, no thyroidism exists. This statement is a good general rule, and we believe that the physician who sees but an occasional case of thyroid disease in his practice, and follows this rule, will make very few errors. In a clinic where large numbers of patients with real and suspected thyroid disease are examined, however, there are found a small group who are, apparently, exceptions to this finding. The diagnosis of this group of borderline cases of thyroid disease is always an interesting problem and frequently a matter of difficulty and uncertainty.

Clinically, cases of borderline hyperthyroidism fall into three groups: In one, the history and physical examination are entirely consistent with hyperthyroidism, yet the basal metabolic rate is normal or subnormal; in the second, neither the history nor the physical examination is typical of hyperthyroidism, yet the basal metabolic rate is elevated; the third consists of a group of neurocirculatory asthenic patients in whom the history and physical findings are both atypical; these can be properly interpreted only after learning the true basal metabolic rate and after repeated clinical examinations.

Since Lugol's solution has been widely adopted for the treatment of hyperthyroidism, many of the usual clinical aspects of the disease have been altered materially.

Today one rarely examines a patient with any kind of a goiter who is not taking or has not been given, iodine. One result of this therapy is that an occasional patient with true exophthalmic goiter will come to the clinic for the first examination in the progress of an iodine remission. Furthermore, spontaneous remissions may occur in the course of the disease. If the patient has had the disease a long time, the history will disclose a typical onset with the usual increased appetite, loss of weight, nervousness, rapid heart, and other characteristic subjective symptoms. The examination may show the residual effects of the thyroidism, such as pigmentation of the skin, exophthalmos, edema of the eyelids, irregular heart, a firm involuted thyroid gland, and possibly emaciation. The basal metabolic rate in such a patient, due to the iodine or to the spontaneous remission, may be either low or within normal limits.

In our opinion, such a patient still has exophthalmic goiter and should have a subtotal thyroidectomy. When we first encountered this type of case, early in our experience with thyroid disease, we frequently refused operation. It was then our firm belief that hyperthyroidism did not exist in the absence of an elevated basal metabolic rate. By that, we meant a rate well above the *average* normal range between +10 or −10. The fact was persistently impressed upon us, however, that these people who had the usual symptoms and signs of exophthalmic goiter were sick and incapacitated even though their basal metabolic rate was normal. Operation was, therefore, undertaken in certain cases and very marked improvement followed.

Check-up metabolism tests after the thyroidectomy often showed the basal metabolic rate to be definitely below the average normal level; yet the patients were clinically well. In a recent study of the end results in 100 cases of exophthalmic

* From the Lahey Clinic, Boston. Read before the American Association for the Study of Goiter, Denver, June 18–20, 1928.

goiter followed for a year or more after operation, it was found that the basal metabolic rate was −10 or below in 19 cases. Only 6 of these patients, however, had any clinical evidence of myxedema.

It is our belief that there are many people who are perfectly well whose basal metabolic rate is commonly below −10 after thyroidectomy. Some observers believe that in these patients the basal metabolic rate in health is low and that a rate of +13 in such a case is equivalent to a marked elevation of metabolism in a patient whose rate is usually ±0. This is similar to Dr. E. P. Joslin's statement that a temperature of 99.2°F. in a severe diabetic is equivalent to a high fever in a healthy person.

In spite of the foregoing, however, it should be emphasized that great care should be exercised in the diagnosis of exophthalmic goiter in patients who have a normal basal metabolic rate. The presence of a soft colloid enlargement of the thyroid in a nervous, excitable, adolescent girl must not be hastily considered evidence of exophthalmic goiter. Repeated examination and tests are necessary in each case and great emphasis must be placed on the presence of the secondary symptoms of hyperthyroidism. Without positive evidence, it is better to withhold operation in these borderline cases. It is, furthermore, our experience that operation in such a case must not be lightly undertaken simply because the basal metabolic rate is normal. Serious postoperative reactions may occur in these cases and the operation must, in certain instances, be done in multiple stages. Each patient must be most carefully prepared for operation in spite of the low metabolism. Moreover, when operating upon this group of patients, it is desirable to leave a larger remnant of each lobe than is customary with the more toxic patients. Failure to do this may result in the occurrence of myxedema after operation.

If the patient who comes for examination during an iodine remission of the disease has had exophthalmic goiter for but a short time, the true diagnosis may be impossible at the first visit. In these cases nothing can replace, in importance, a carefully taken history with particular emphasis on onset, weight changes, appetite, menstrual history, muscle weakness and heart rate. The examination may disclose little positive evidence of hyperthyroidism other than a thyroid gland which is firm in consistency and slightly enlarged. Iodine treatment often causes the exophthalmos and stare to disappear, the tremor to vanish and the heart rate to return to normal. Furthermore, in these cases, the basal metabolic rate is often within normal limits or but slightly elevated. In certain patients in this group we may suspect hyperthyroidism in a remission, but, lacking sufficient positive evidence for the diagnosis, it is our custom to send them home for a period of six weeks of normal activity without medication of any kind. At the end of this interval they return for further examination and for further metabolism studies. If the metabolism is still normal and the symptoms are improved after this interval, we believe we must classify them as patients with no thyroid disease and refuse operation. If, however, they return after this interval with an exaggeration of their previous symptoms and an elevated metabolism, operation is advised.

The second group of patients whom we may term border-line is that group in which the symptoms and signs of the disease are decidedly atypical, yet on repeated examination the basal metabolic rate is elevated. The leading symptom in this group of patients varies greatly. "Nervousness" is perhaps the most frequently encountered, but loss of weight, trembling of the extremities, or palpitation not infrequently bring the patient for medical advice. Frequently basal metabolism tests are done on patients as part of a complete physical examination. Occasionally these tests are found to be high. It is then necessary to determine the significance of the high basal metabolic rate.

It is hardly necessary to stress at this time the importance of a most careful control of the actual technique of the test. We not infrequently examine patients whose metabolism is reported as very high, but whose true basal metabolic rate, after well-controlled or repeated tests, is found to be within normal limits. Certainly in every questionable case the test must be repeatedly taken and carefully checked before it is accepted.

It is, of course, well recognized that the basal metabolic rate is frequently elevated in lymphatic leucemia, polycythemia and pernicious anemia. In any unusual case presenting a high rate, the possible presence of one of these blood diseases may be settled at once by appropriate blood studies. We have noted certain patients with marked chronic nephritis and hypertension who constantly showed a basal rate of +40 or more. These cases are very difficult diagnostic problems and may easily be operated upon with the mistaken diagnosis of hyperthyroidism. It has been our experience that subtotal thyroidectomy fails completely to benefit the symptoms due to hypertension in these cases and that the basal metabolism persists at the same level after thyroidectomy. We have studied one such patient with hypertension who had a basal rate of +40 which varied very little with rest in bed and iodine. Subtotal thyroidectomy produced no change in either her symptoms or her basal metabolic rate. A year after operation she died of congestive heart failure and at autopsy showed no other cause for her difficulties than arteriosclerosis and chronic nephritis.

The correct diagnosis of many of these cases will finally depend on a most careful consideration of the history of the illness and a special search for any secondary symptoms of hyperthyroidism. We may consider exophthalmos, tachycardia, tremor, goiter, and an elevated metabolism as primary and typical evidence of hyperthyroidism. These findings are practically pathognomonic of exophthalmic goiter. In all exophthalmic goiter patients, however, careful questioning and examination disclose numerous other symptoms arising as a result of and in addition to these primary findings which we consider of secondary importance in determining the diagnosis. No one of these secondary symptoms is pathognomonic of exophthalmic goiter; but the presence of several of these symptoms and signs in a patient is frequently of definite diagnostic value. In all borderline cases, therefore, where the primary evidence of hyperthyroidism is absent, the secondary symptoms of hyperthyroidism become of great diagnostic importance.

Unaccountable variations of marked degree in the patient's weight in the absence of any other cause are very suggestive of hyperthyroidism. It has been our experience that a patient losing 20 or more pounds in spite of an exceptionally good appetite usually has either diabetes or hyperthyroidism. The fact that a trace of sugar is present in such a patient's urine does not settle the diagnosis, since it is very common to find a trace of sugar in the urine in hyperthyroidism. A fasting blood sugar and careful basal metabolic studies must be done to establish the correct diagnosis.

A sensation of increased body warmth is a valuable secondary sign in hyperthyroidism. Accompanying this are increased sweating and vasomotor instability, especially shown in the skin of the chest and neck. A diminution in the menstrual flow is commonly noted after the onset of hyperthyroidism. Complete amenorrhea may occur. Such changes in the menstrual flow become of distinct diagnostic importance in the patient suspected of having hyperthyroidism when considered with other findings. Attacks of diarrhea with or without vomiting, in the absence of any other cause, suggest a milder degree of the increased intestinal activity seen in the acute crises of hyperthyroidism.

Of less diagnostic value are the subjective nervous symptoms of the disease.

These are of importance only as the history notes their occurrence definitely with the onset of the present trouble. Thus it becomes significant if a person previously quiet and mentally composed develops marked emotional instability, passing from laughter to tears without adequate cause. Marked irritability and inability to put up with the trivial annoyances of life when developing in a patient previously amiable are suggestive of the presence of thyroidism.

Possibly the most important of these secondary evidences of hyperthyroidism is the evidence of muscle weakness. It is generally known that marked muscle weakness accompanies severe hyperthyroidism, but the diagnostic importance of this finding in the borderline cases is often overlooked. In many instances the history is specific on this point and states that the patient noted a marked loss of muscle power in legs or arms and that the knees became weak and often "gave away."

More often, however, the evidence of muscle weakness can only be obtained by some direct test of muscle strength. For this purpose we have found the quadriceps test devised by Dr. F. H. Lahey of distinct value. The patient while seated well forward on a chair raises one leg to a horizontal position with the knee extended. A marked strain is exerted upon the quadriceps extensor tendon and considerable muscular effort is required to sustain this position. The length of time that a normal person can maintain this position varies from forty seconds to two minutes or more. It is extremely rare to find a patient with any degree of thyroidism who can hold this position for more than thirty seconds. Occasional cases of undoubted hyperthyroidism may have no marked muscle weakness. A low quadriceps test, however, is valuable evidence of hyperthyroidism in the presence of other suggestive findings.

The diagnosis of the borderline hyperthyroidism patient who lacks the primary or usual evidences of the disease, but shows a high basal metabolic rate, will be dependent very largely on the history and secondary evidences of hyperthyroidism.

The differential diagnosis of neurocirculatory asthenia from early or borderline hyperthyroidism is in many instances dependent on long experience and observation. There is, furthermore, no patient for whom a thyroidectomy will do as little good or more harm than the neurocirculatory asthenic. One must, therefore, be constantly alert that he does not overthrow the already poorly balanced nervous system of this type of patient by an unnecessary thyroid operation.

Many of the symptoms of hyperthyroidism are commonly present in the neurasthenic and on this account must not receive undue emphasis in diagnosing the borderline case. Thus tremor, nervousness, palpitation, and rapid heart are leading symptoms consistent with either diagnosis. The mental outlook of the two diseases is vastly different. The neurasthenic is pessimistic and fearful of the future. The hyperthyroid patient is optimistic of her ability and hopeful of the future. The neurasthenic feels that she cannot do her daily work, but does it. The hyperthyroid patient feels sure she can perform most tasks, but after trial, fails.

Alteration in the menstrual function is rarely present in the neurocirculatory asthenic patient. Diminution or cessation of the menses is common in the toxic goiter case. The neurasthenic is plump and healthy in appearance, but says she can scarcely eat a thing. The toxic goiter patient usually has an excellent or excessive appetite, but nevertheless is thin or is losing weight.

The presence of a goiter is, in this type of patient, of course, most suggestive of hyperthyroidism. It does not by any means, however, confirm that diagnosis. We have repeatedly seen a moderate thyroid enlargement accompanied by symptoms of neurocirculatory asthenia

which were so confusing that they could only be interpreted properly when the basal metabolic rate had been found to be normal.

The basal metabolic rate is undoubtedly the only measure at present which will permit us to detect many of these cases of neurocirculatory asthenia. In any suspicious case the rate must be taken several days in succession before it becomes certain that the true rate has been obtained. It is our belief that in certain cases a rate a few points above the average figure may be just as normal for an individual as a rate a few points below the average normal figure. In the last analysis, the diagnosis in certain very doubtful cases will rest largely on the observer's impression of the patient and his experience with similar cases. When the diagnosis is as doubtful as this, it is usually the part of wisdom and good judgment to postpone the decision for or against operation until the time when more positive evidence of hyperthyroidism is present.

Exophthalmic goiter patients who have had a thyroidectomy occasionally return for examination because they fear their disease is again becoming active. This group of patients not infrequently presents unusual features that are worthy of note.

There are many of these patients who are chiefly troubled with worries rather than symptoms. They are fearful that the disease may be returning, although they have had no change in their health. Others of this group have slight pains in their neck or are attacked by "nervous spells" at frequent intervals which they believe point to a return of the goiter. Rarely one encounters a patient who is obviously a severe neurasthenic and who reports a group of symptoms which reveal imaginary trouble in all parts of the body, but in no way point to any thyroid disorder. Such a case can generally be readily established as a neurasthenic and one may wonder if hyperthyroidism had been present before the thyroidectomy. Certainly an occasional visitor of this type will do much to deter the surgeon from early operation in borderline cases of hyperthyroidism.

There are, of course, certain patients who have very definite hyperthyroidism at varying periods of time after subtotal thyroidectomy. Some of this group are frankly toxic and the diagnosis is obvious, while others are so mildly toxic that many are borderline cases and require careful diagnostic study.

Some time ago, Dr. F. H. Lahey and the writer[1] called attention to the fact that hyperthyroidism present after thyroidectomy for exophthalmic goiter might be a true recurrence of the disease, but that in the majority of cases it was simply a persistence of the disease. The only difference between persistent and recurrent postoperative hyperthyroidism is in the time of their onset.

Persistent postoperative hyperthyroidism results from the surgeon's failure to remove enough hyperplastic thyroid tissue. Failure to remove sufficient tissue in any given case is most frequently due to the inexperience of the operator, to unusual technical difficulties in the operation from hemorrhage, or intense intoxication, or to improper judgment as to the degree of secretory activity inherent in the thyroid tissue left in the neck.

Since the last two possibilities are in a large measure unavoidable and occasional cases of persistent hyperthyroidism may occur, it has long been our custom to insist on repeated metabolism tests and clinical examinations of all toxic goiter patients after operation. We expect to find no clinical evidence of hyperthyroidism and a normal basal metabolic rate at the first examination three months after operation. If the basal metabolic rate is high at this time and there is still evidence of hyperthyroidism, we give the patient Lugol's solution M.X daily and ask her to return for another examination in six weeks instead of waiting the usual

[1] Lahey, F. H., and Clute, H. M. Persistent and recurrent hyperthyroidism. *Ann. Surg.*, 83: 199, 1926.

three months for the next test. If the test is high and there is still evidence of persisting hyperthyroidism at this time, further removal of thyroid tissue is urged.

The apparent absence of large remnants of thyroid tissue in such a case should not delay the advice for further operation. True persisting hyperthyroidism is caused by the overactivity of the thyroid remnants, whether they are large or small, and is cured by their removal. We have rarely, if ever, failed to find large thyroid remnants of hyperplastic tissue in any case of persistent hyperthyroidism that we have submitted to a second operation.

Occasional cases have been seen in which the patient's symptoms of persistent hyperthyroidism were completely relieved by the regular administration of Lugol's solution, but this group is quite small. These cases have shown neither high basal metabolic rates nor large thyroid remnants.

True recurrence of hyperthyroidism after a prolonged period of normal health may occur, but it is certainly unusual. Here again it is important to recognize the fundamental fact that the hyperthyroidism is due to an excess of hyperfunctioning thyroid tissue, and that it can be cured only by the removal of a large part of that tissue. Lugol's solution in these cases, as in any other case of primary hyperthyroidism, should be used only as a means of preparing the patient for operation, and should not be expected to produce a permanent cure unaided by surgery.

It is our belief that in either persistent or recurrent hyperthyroidism, further removal of thyroid tissue must usually be undertaken. The earlier this decision can be reached in any case, the better. We would much prefer to have a patient in need of daily thyroid medication than to have her constantly under the drive of a hyperfunctioning thyroid gland.

THE IODINE QUESTION IN ANIMAL EXPERIMENTS*

D. B. BREITNER

VIENNA, AUSTRIA

THE subject I have taken the liberty of choosing is the question of iodine in experiments upon animals. But it is necessary that I first make some general remarks. Nowadays it is impossible to approach the subject of goiter from any point without it becoming a whole book. We stand on the threshold of a new orientation in the question of goiter that can never be overlooked. This new orientation extends over all the details of the teaching of the diseases of the thyroid gland.

It seems to me worth noticing that this new orientation consists, not in the giving of our present knowledge, but in the recognition of a connection which we have not known until now. In this way we have won a new basis for the diagnosis, prognosis and treatment of the diseases of the thyroid gland.

The International Conference in Berne in August, 1927, was very interesting and important. I confidently hope that the American Congress in Denver will become the starting point of a new teaching on the diseases of the thyroid gland. For we must agree upon some cardinal points.

When I say, "This man has a goiter," the trouble starts with the word goiter. I say no more than if I said, for example, "This man limps." But that is a very modest diagnosis. Why does the man limp? Has he coxitis? Has he a badly healed fracture? Ischias? Perhaps a wooden leg? To this question, "Why," the physician must give the answer, when even the children in the street can make the diagnosis "the man limps."

Why has this man a tumor on his neck, which tumor belongs to the thyroid gland? That is the first question.

We may find that the man has carcinoma of the thyroid gland, sarcoma, dermoid, echinococcus, inflammation of the gland. All that does not belong to the problem of the thyroid gland. But if we find a diffused symmetrical swelling of the whole gland without any of the above mentioned diseases then it is an alteration of the gland which is characteristic of it as an endocrine organ. This alteration therefore, must have a connection with the function of the organ. That is the first step in the new teaching about the diseases of the thyroid gland.

The diffused alterations of the whole organ, and only this we shall call for the present by the name goiter, are the expressions of functional disturbance of the organ.

The second step must make the decision whether these functional changes are primary or secondary disturbances of the normal functions of the gland. To this thought we must give a short consideration.

There is an endemic, an epidemic, a sporadic goiter. The endemic and epidemic goiter have a decided histological character. The sporadic goiter can show different histological structures, but also those of the endemic and epidemic goiter.

It may be that in all these cases a certain malignancy is working in the gland and leads to the same injury. If that is so the goiter would be a primary disease of the thyroid gland and a certain histological picture belongs to this disease. But here an important factor comes into consideration. It is possible in experiments on animals to produce these histological changes which are characteristic of spontaneous goiter. From that we can see that these changes can be possible under different conditions. So they are not a

* Read before the American Association for the Study of Goiter, Denver, June 18–20, 1928.

primary specific disease of the gland but much more the expression of a changed function through the influence of different causes.

Therefore the second diagnosis is: The functional diseases of the thyroid gland have a conditional nature, or in other words, the goiter is a symptom. As for the question of etiology, this diagnosis is the end of the dispute about uniform etiology. Different causes can be effective at the same time. So these causes can make the histological changes an expression of the same functional change. The dispute about the water theory, the iodine theory, the dirt theory, the infection theory and all the other theories loses its monopoly.

Not one alone, but everyone is right in some respects, because completely different conditions can lead to the same functional changes.

It is easy to understand what a great change this comprehension makes in the diagnosis, prognosis, indication and in the kind of treatment.

The solution of all these problems lay in the utilization of experiments on animals.

If I now come to the real subject of my address, I must point out how *little* the experiments on animals have been made use of in solving the questions.

Hurtle went along this road but he left it again directly. America brought the first results of systematic experiments on animals.

It is owing to Crile, Marine and Lenhart that the first deep insight was made into the morphological basis of the physiology of the thyroid gland. To these, further experiments of de Ligneris were joined, which were successfully continued by de Quervain and his pupils.

Sixteen years ago I tried to build up again the whole structure of the physiology and the pathological—physiology of the thyroid gland based on the experiments on animals. One part of the results of these experiments, the question of iodine, I have the honor to place before you now.

You know very well the opinion of physiologists about the importance of iodine for the thyroid gland and also the interesting history of the treatment with iodine.

Again to America belongs the merit of having taken the decisive step in this most important question. The works of Wilson, Plummer, Boothby, Scott and others have reached the highest point in the teaching of the therapy of the thyroid diseases.

The iodine therapy of hyperthyroidism was already known to Basedow and Trousseau. First with deliberation it was performed by the German authors, Neisser, Loioy, and Zondek. In a large style America used this therapy.

Iodine, that remedy so much feared in goiter, that three times feared medicine in hyperfunctional conditions of the gland, has just under these conditions become a miraculous remedy!

This is such a revolutionary and also such an incomprehensible fact that the explanation by means of experiments on animals seemed to be a useful work. Let us review these experiments.

If we remove half the thyroid gland of a normal dog, in the course of weeks and months we shall find that the other half grows larger. This is only the confirmation by experiment of old clinical experience.

If we look at the remains of the gland some months later we shall see a normal histological picture; that means we shall find a eutrophic view of the cellules, to use Aschoff's expression and a change of the colloid contents. But if we remove the second part of the gland only a few days later, and if we observe the histological structure of this part, then we shall see a distinct difference between that and the first half that we removed. In the second part no colloid is to be seen.

But if we give the animal thyroid substances with his food beginning from the day of the removal of the first part, the histological view is quite the same; there is again colloid in the gland.

That is a fundamental experiment. Let us make a new one. We shall make a

stricture of the trachea of a normal dog with a silk thread. Then we make a trial excision of the gland; some days later we remove the gland and examine it histologically. We shall see that the follicles are quite full of colloid. This second fundamental experiment was first made by the two German surgeons Blauel and Reich. So we can bring out by the first order of the experiment, diminution of colloid, by the second arrangement, increase of colloid in the gland of the animal.

If we now make both arrangements at the same time, that means, if we make a stricture of the trachea and a resection of the gland, the histological picture remains almost normal. To create a better understanding I have described the condition of colloid *diminution* as "hyperrhoe" and that of colloid *increase* as "hyporhoe."

If we try to bring to one formula these effects of the experiments we can say: The quantity of colloid stands in relation to the need of secretion in the organism.

Increased flow of secretion leads to diminution of colloid in the gland, obstructed flow leads to increase of colloid. We must consider the colloid as a reserve material of the gland.

Now what happens when iodine is given to the animal? If we give iodine to that animal which is obliged to retain colloid by the stricture of the trachea, then the hyporhoe is not to be seen.

If we give iodine to the animal that is obliged to reject the colloid by a resection of the gland then the hyperrhoe is not to be seen. Therefore in every case iodine works against the functional direction, *entgegen der funktionellen Richtung*, of the gland.

These are the results of experiments on animals. It is amazing how simply these experiments explain clinical observation!

You all know the picture of iodine therapy. A patient with a diffused symmetrical goiter, that means a functional one, is ordered to take iodine. Some time later symptoms appear which are very similar to those of Basedow's or Graves' disease. The goiter becomes smaller. But the symptoms which we understand as hyperthyroidism, can reach a very disquieting point. And now the remarkable thing occurs: The iodine is stopped by the physician, the symptoms of hyperthyroidism diminish, and the goiter returns. What happened? The hyporhoic goiter has been mobilized. As in the experiments on animals, the iodine worked against the functional direction which had the tendency to retain the colloid. In the organism, whose need of secretion made the hyporhoe, this retention of colloid is overcome by force. The organism responds with the symptoms of the overloading of secretion.

But as soon as the supply of iodine is stopped the original functional direction of the gland appears again; it again retains colloid, the symptoms of hyperthyroidism disappear, the goiter increases. And now we take the contrary. By a physical trauma, by an endocrine disturbance, a gland is brought into the state of hyperrhoe. The clinical view of this state is hyperthyroidism, Graves' disease. Now this patient gets iodine. In a few days the acute symptoms of hyperthyroidism disappear. But if we stop the supply of iodine, some days later the old symptoms return with renewed violence. What has happened now? Just as in the animal experiments the functional direction of the gland, which was hyperrhoe has been changed by iodine to the contrary. By that the excess of secretion was stopped, the hyperthyroid symptoms disappeared.

But as soon as the supply of iodine is stopped, the original functional direction gets the upper hand again, the retained secretion is washed away, the hyperthyroid symptoms appear again with renewed violence. A long time ago the histological examination of human goiter proved the correctness of this opinion.

Already in 1927, the two Americans, Giodarno and Caylor,[1] showed that the

[1] Giodarno and Cayler. *Surg. Gynec. & Obst.*, 1927.

amelioration of the hyperthyroid symptoms appear at the same time as the increase of colloid in the gland. Since then the works of Rienhoff, Jr., of Baltimore, Schurer of Vienna, Merke of Basle have given incontestable confirmation of this fact. Following the method of Boothby and Plummer, supplying iodine to patients with hyperthyroidism, causes an increase of colloid in the gland, which proceeds simultaneously with the amelioration of the clinical symptoms. That is the effect of iodine in hyperthyroidism, that is the essence of this bold and ingenious idea.

My report of the iodine question reaches as far as this in animal experiments; and, it is a matter of course that, with all this, not all the details of clinical observation are thoroughly cleared up. But the experiments on animals should serve to find out a broad line. The jungle of problems in the pathology of the thyroid gland should receive at least one path. Then the farmer must come and make the road passable and do the other things that belong to the work of the farmer.

But you must allow me just another word, because for all facts we must think out a reason. We can only believe that the effect of iodine lies in the influence of the secreting nerve of the gland.

We owe to the pharmacologist H. H. Mayer and to the internist Jannuschka of Vienna an interesting confirmation. Iodine can work in an organism as a sedative or as a stimulant. When in an organic system, under normal conditions, the sympathetic and parasympathetic systems are well balanced, then the functional disturbances appear by the overweight of one part of the system. In the thyroid gland the irritation of the sympathetic system leads, as Susani of Vienna shows, to hyperrhoe, irritation of the parasympathetic system to hyporhoe, now iodine always works in the direction of the normal condition.

In our case the iodine calms at once the increased tonus of the sympathetic system, by which the hyperrhoe is stopped.

With this effect of iodine on the different forms of goiter and on its clinical symptoms, the condition is brought nearer to our understanding. It is easy to consider the whole pathology and pathological physiology from this point of view. A new but perfectly enclosed and logical system is the result.

The particular forms of struma do not stand next to one another, without contact and quite strange to each other. Everything is a living chain of reversible proceedings but not a cabinet of minerals all neatly labelled!

We see the functional types of goiter as symptoms of disturbances of the whole organism. The reason for these disturbances can be exogenous or endogenous. We see the single goiter as a distinct functional phase which we diagnose before we have the preparation in our hand.

We see the prognosis of the single case in the light of its functional nature and take our therapeutic measures accordingly, and we meet the old specter of relapse with a sure hand because we know his nature. The appearance of the goiter of puberty, the adolescent goiter, the struma premenstrualis goideuhain, the goiter during the time of pregnancy and climacteric Basedow, all these are to be understood in their substance by the new method.

We understand a biography of the normal gland in the same way as a pathography of the altered gland. From these two we form a biology of the organ so that this organ with all the qualities of its life is in the grasp of our understanding.

I do not need to lay stress upon the fact that this system too has its limitations, as has all knowledge of natural science.

But the aim and end of our work is to make all our facts accessible and useful.

It is very agreeable to me to lay stress upon the important pioneer work done by the American Research Society on the solution of goiter problems. And at the end of an address about animal experiments in goiter studies, I may point with special pleasure to George W. Crile, the father of these experiments.

THYROIDECTOMY IN THE MENTALLY DISTURBED WITH EXOPHTHALMIC GOITER

WITH REPORT OF TWELVE CASES IN WHICH THE PSYCHOSIS WAS RELIEVED BY OPERATION*

JOSEPH L. DECOURCY, M.D.

CINCINNATI

THE mental condition of patients with exophthalmic goiter is, as a rule, far from normal; yet we seldom associate the presence of frank psychoses with diseases of the thyroid gland. It is well known, however, that persons with Graves' disease are generally emotionally "on edge;" that they are easily startled, annoyed, or brought to tears or anger; and that they are likely to suffer from insomnia and spells of depression or excitement.

During the last five years, my experience with goiter surgery in mentally unbalanced patients has led me to believe that, when a frank psychosis coexists with exophthalmic goiter, thyroidectomy offers a fair prospect of relieving the mental disturbance. I have had occasion to operate upon 14 insane patients with Graves' disease, and all but 2 of them made complete mental recoveries and have remained normal to date. To this work I shall refer later.

Twenty years ago Berkley and Follis[1] observed a marked resemblance between the symptoms of exophthalmic goiter and those of the catatonic form of dementia precox. In their study of the mental symptoms occurring during the prodromal stage of catatonia, they were much impressed by the similarity between them and the mental peculiarities of patients with Graves' disease. They pointed out that in the somatic realm tachycardia, rapid loss of weight, excessive sweating, a fine tremor in the fingers, quick pupillary reflexes, vasomotor weakness and menstrual anomalies are common symptoms in both exophthalmic goiter and catatonia.

The thyroid gland itself was observed to show rather constant changes in cases of catatonia. Although it was generally not enlarged, it was usually of a peculiar soft and mushy consistency and contained occasional hard nodules. When iodine, iodothyrine or desiccated thyroid extract was administered to patients with dementia precox, they came out of their stupor temporarily.

Berkley and Follis performed partial thyroidectomy in 10 cases of catatonia. As a result, 4 of the patients recovered their mental equilibrium, 3 of them improving slowly but progressively and showing no tendency to relapse.

Other investigators have confirmed the work of Berkley and Follis, so that there can be no question but that, in occasional cases, there is a close relationship between dementia precox and thyroid disease. Of course, it must be distinctly understood that no attempt is being made to assign an etiological relationship of the thyroid gland to the average case of dementia precox; for the mass of evidence is opposed to this view.

Kanavel[2] observed definite improvement but not complete cure in a case of dementia precox of recent standing in which he removed the thyroid gland. However, in 9 cases of long standing there was no mental improvement following the operation.

* Read before the American Association for the Study of Goiter, Denver, June 18–20, 1928.

Winslow,[3] in a series of 5 cases of dementia precox in which partial thyroidectomy was performed, noted much improvement in 3; but 2 of the patients later relapsed. He concluded that there is apparently some relation between the thyroid gland and catatonia; that partial thyroidectomy frequently confers great benefit, when the operation is performed in the early stage of the disease; but that thyroidectomy late in demetia precox is without benefit.

Weinberg[4] noted complete relief of the mental symptoms after thyroidectomy in a case of dementia precox and one of manic-depressive psychosis, both with typical symptoms of exophthalmic goiter. Eastman and Eastman[5] performed hemithyroidectomy in 4 cases of the paranoid form of dementia precox in which one lobe of the thyroid gland or one lobe with the isthmus was enlarged. In each case the psychosis was entirely relieved. Jackson[6] observed recovery after operation in a mentally disturbed woman with typical symptoms of exophthalmic goiter. Boys[7] reported 8 cases of goiter associated with well-developed mental symptoms, in 6 of which thyroidectomy resulted in complete recovery.

My own experience with thyroidectomy in cases of combined exophthalmic goiter and mental disease has been exceedingly favorable. As I have already stated, 12 of 14 frankly insane patients upon whom I operated for Graves' disease during the last five years made complete mental recoveries and have retained a normal mental status to date. Two of these patients were young subjects, in whose cases diagnoses of dementia precox had been made and poor prognoses given; yet both recovered. In 13 cases the condition of the thyroid gland was hyperplastic goiter; in 1 toxic adenoma also with some evidence of hyperplasia.

I should like to mention 2 of my cases as typical of those with favorable results in this series. I saw a woman, thirty-five years old, at the Connecticut State Insane Asylum, Middletown, Conn., who had lost 40 lbs. during the preceding six months. Her pulse was very weak, ranging between 140 and 160. The outstretched fingers showed a very pronounced fine and rapid tremor. There was an adenoma about the size of an egg over the right lobe of the thyroid gland, which was enlarged and of the consistency usually found in hyperplastic goiter.

During the journey to Cincinnati, the patient kept muttering unintelligibly and made several attempts to leave the train. On several occasions she tried to swallow her own feces. She improved physically under rest and Lugol's solution, but her mental condition remained the same. Thyroidectomy was performed and the postoperative course was uneventful. At the end of a month her weight had increased and her pulse rate was normal. Later her mind began to clear and three months after the operation she appeared mentally normal. A month later her mental condition was so satisfactory that she was allowed to make a motor trip East by herself. She has now been under observation for a year and is entirely well and there is every reason to believe that she will remain so. I believe that her mental disturbance was of the nature of an exhaustion psychosis and that thyroidectomy, by eliminating the source of toxemia, played a large part in restoring mental balance.

The second patient was a married woman, thirty years old, whose mental symptoms suggested the paranoid form of dementia precox. At times her behavior was very strange. Once she left home and was found late that night wandering aimlessly in a nearby woods. She refused all forms of medication, particularly when her husband tried to give it to her, because she imagined that an attempt was being made to poison her. She had a number of delusions of persecution, but they were in the main poorly organized, as is characteristic of the paranoid form of dementia precox.

For four years she had noticed that her

neck was unusually full. During the previous year the swelling had increased. At the same time, she became more irritable and emotional, until finally her conduct began to appear decidedly abnormal. Her last child was born fourteen months previously, and she had lost 20 lbs. since that time.

The appearance of the patient was typical of thyrotoxicosis. She was excited, apprehensive and considerably under weight. Her pulse rate ranged between 130 and 140, and the thyroid gland was increased to about three times normal size and was of the consistency found in hyperplastic goiter.

During the first week after thyroidectomy, which was performed after the usual course of preparation with Lugol's solution, the mental symptoms became worse. Frequently the patient became so excited and uncontrollable that hypnotics were required. Twice she ran away from home. Three months after operation, however, it was apparent that gradual improvement was on the way. At the end of a year, during which improvement was slow but steady, the patient appeared entirely well and her mental condition was clear in every respect. She has remained well both mentally and physically, and there is no reason to believe that she will relapse.

In 10 other mentally disturbed patients upon whom I operated for exophthalmic goiter the mental symptoms finally disappeared after thyroidectomy. In 2 cases there was no benefit from the operation. One of these patients died in an asylum and the other is still an inmate.

Experience in the treatment of mental diseases has proved that anything which improves the general health of the body increases the likelihood of recovery from the psychosis. Exophthalmic goiter is in itself a cause of much nervous instability and unrest. Given a person of the schizophrenic type, it may well serve as an exciting cause of mental breakdown. I do not believe, however, that exophthalmic goiter is related to dementia precox or other psychoses, other than as a precipitating and contributory cause.

In performing thyroidectomy on patients with psychoses, it is doubly important to insist upon preoperative rest and treatment with Lugol's solution, because of the danger of increasing the mental symptoms by a period of exacerbation that might otherwise follow immediately after the operation. Furthermore, in patients with mild psychoses, we must be prepared to exercise restraint, if necessary. Mental betterment seldom occurs immediately after thyroidectomy but is more commonly of gradual evolution during a period of from several months to a year.

In conclusion, I should like to emphasize the fact that the coexistence of mental disease with exophthalmic goiter does not constitute a contraindication to operation but rather makes thyroidectomy doubly advisable, since it offers a fair prospect of mental recovery.

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THE TREATMENT OF THE DESPERATE GOITER PATIENT*

J. EARL ELSE, M.D.

PORTLAND, OREGON.

THE mortality and morbidity following operations for toxic goiter are now fairly good in patients with moderately toxic goiter showing no extraordinary symptoms or pathology. The great difference between the mortality and morbidity rate of surgeons obtaining the best results and those obtaining only mediocre results is usually in the so-called desperate goiter patients. This paper will discuss the following: 1, severe hyperthyroidism without crisis; 2, hyperthyroidism with crisis; 3, hyperthyroidism with fibrillation; 4, hyperthyroidism with extreme hypertension; 5, hyperthyroidism in pregnancy.

1. Severe hyperthyroidism without crisis. It would seem that with the use of Lugol's solution as suggested by Plummer and of the preparation of the patient for operation as emphasized by Crile, the mortality should be low, yet it is not as low as it should be. The chief cause of mortality and morbidity in this group is lack of preparation before operation. The patients are operated upon before they are ready and death is the result. The master goiter surgeon may be able to judge when the patient is ready for operation without laboratory assistance, but the mass of surgeons operating upon goiter cannot, and death often results in their attempt to imitate the master by disregarding such aids. While the master goiter surgeon by his skill may be able to tell when the severely toxic patient is ready for operation, he cannot by his skill so accurately distinguish between the mildly toxic goiter and other conditions which so closely simulate it that only the basal metabolic rate will differentiate. Neither can he by his skill so accurately distinguish between the patient requiring stage operations and the one who does not, so he treats all as severe cases. There is no surgeon so skilled that he can differentiate as well without laboratory aids as with them, and the surgeon who does not use laboratory aids either subjects some of his patients to unnecessary delay with unnecessary stage operations, or has an operative death-rate higher than it should be.

Patients with severe hyperthyroidism should be sent to the hospital for preparation. It cannot be done well at home. Patients should not be permitted to get out of bed for anything or consulted about any matters pertaining to business or other responsibilities. Lugol's solution should be given in amounts varying from 10 minims three times a day to 25 minims four times a day according to the severity of the disease. Nervousness should be controlled by giving 5 to 10 grains of veronal in hot milk every night at 9 o'clock as routine, and additional veronal or pantopon if needed to make the patient perfectly contented. The diet should be watched, with selection of food that the patient likes and can digest easily, and eggnog, milk or chocolate should be given between meals. We make it an absolute rule never to do a radical operation on such a patient with a basal metabolic rate over plus 50; under plus 40 is preferable and even then we do not operate until the patient's symptoms show a very definite improvement. Further, it is an absolute rule never to operate upon such a patient if he is apprehensive of the result. I do not believe in the so-called stealing of the goiter. With the judicious use of sedatives, Lugol's solution, a carefully forced diet

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and proper nursing, these patients instead of being apprehensive are anxious for the day of operation to come. In the occasional patient who cannot be satisfactorily prepared by this method, the stage operation is indicated but as time goes on and experience becomes wider, we find fewer patients in this group who require the operation in stages, so that now it is a rare thing for us to do a ligation of the superior pole and almost as rare to remove the lobes one at a time.

2. Hyperthyroidism with crisis. Crisis occurring in severe hyperthyroidism is probably due to an acidosis. The markedly increased catabolism necessitates increased excretion and an increased dissipation of heat. The blood supply of the skin is increased in order to hasten the dissipation of heat through radiation and evaporation. The rapid evaporation reduces the fluids of the body concentrating the solids of the blood stream and making their absorption from the tissues more difficult. The increased blood supply to the skin means a relatively decreased blood supply to the kidneys, consequently, these patients are prone to develop an acidosis. Until we began to use glucose intravenously we had a limited number of patients each year coming in with an acute hyperthyroidism with vomiting and diarrhea which we were unable to control, and consequently, we were unable to give Lugol's solution because it was immediately expelled when given by either mouth or rectum. These patients usually died within two to four days after entering the hospital. Since we began to use glucose intravenously, I have not seen a single death from this cause. With the exception of patients brought in in a morabund condition this type of death should be extremely infrequent. It is our custom in this group to give patients 1000 c.c. of a 10 per cent solution of glucose in physiologic salt solution intravenously as soon as they enter the hospital. This is given slowly, taking at least an hour for administration and longer if there is evidence of myocardial weakening. Fifteen units of insulin are given at the beginning of the injection of the glucose and the same amount at the conclusion. The vomiting and diarrhea are rapidly checked and the patients are then able to take Lugol's solution by mouth. We prefer giving the Lugol's solution in orange juice or milk in 25 minim doses four times a day. After checking the crisis, the preparation of these patients does not differ from that of those with severe hyperthyroidism without crisis except that it usually takes a little longer to get them ready for operation.

3. Hyperthyroidism with cardiac fibrillation. This group gives us more anxiety than any other group of goiter patients and yet our results are as satisfactory so far as operation is concerned. I have had only 1 death to be termed an operative death in this group and that in a woman seventy-nine years of age. Other patients have later died of heart lesions but this did not occur for some time after leaving the hospital with the exception of 1 patient who died from emboli one month after leaving the hospital.

These patients are put at absolute rest in bed. Veronal and pantopon are used in sufficient dosage to keep them perfectly contented. Lugol's solution is given as to patients with severe hyperthyroidism. The heart condition has been treated by my associate, Dr. Luther T. Nelson, with quinidine, or quinidine and digitalis. Some of the milder cases yield with this treatment and the heart stops fibrillating, but in some of the more severe cases, the heart continues to fibrillate until the final subtotal lobectomy.

It is in this group that the stage operation is most valuable. Upon those patients in whom the heart does not yield, the operation is done in four stages, ligating the superior poles one at a time and doing the subtotal lobectomies one at a time. I have many times observed that while the patient showed definite improvement after each of the stage operations, the heart continued to fibrillate until the

removal of the last lobe. This is indicative of the extent of the operation required. Practically all the diseased thyroid tissue must be removed and new thyroid tissue developed in order to get a normal secretion. Even though the heart may appear to be normal, the patient should be warned that a badly damaged heart probably never becomes completely normal and consequently should never be severely taxed.

4. Hyperthyroidism with hypertension. Hypertension occurs in the course of hyperthyroidism from toxic adenoma or toxic diffuse adenomatous goiter and after repeated attacks of toxic hyperplastic (exophthalmic) goiter. Patients with pronounced hypertension have given me a great deal of anxiety for fear a cerebral hemorrhage would occur as a result of the operation. Up to the present, I have never seen it follow goiter operations, although I saw it only last year following a prostatectomy that was necessary in the presence of high hypertension because of marked urinary retention.

It is our rule never to operate in the presence of high hypertension except in case of necessity. I regard operation in toxic goiter with hypertension as a necessity because these patients gradually get worse. They are given bed rest with sedatives and Lugol's solution for one to two weeks previous to the operation. Ligations are rarely done, as the type of goiter producing hypertension usually does not give definite results from ligating the superior poles. Subtotal lobectomies are done in one or two stages according to the severity of the goiter and the height of the hypertension. Following the operation the hypertension usually is reduced but rarely returns to normal because of permanent damage done before operation.

5. Hyperthyroidism in the latter months of pregnancy. In the early months pregnancy does not constitute a contraindication for an operation for goiter. In fact, I have regarded it as an indication for an immediate operation because the patient with a mild hyperthyroidism may develop a very extreme hyperthyroidism in the course of the pregnancy. I have never seen an abortion occur as the result of the operation on a pregnant woman. In the latter months of pregnancy there is an added risk and I believe it is best to treat these patients with roentgen ray, telling them that it is a temporary measure. While it relieves the attack, it does not cure the goiter, which will recur later in a large proportion of the cases. It is my custom to give these patients Lugol's solution, the amount varying from 10 minims three times a day to 25 minims four times a day for two weeks. Sedatives are given to control the nervous state of the patient and each lobe and the isthmus are given 90 milliampere-minutes of roentgen ray, 3 milliamperes of current for thirty minutes, with a 9 inch back-up, filtered through 6 mm. of aluminum and ½ inch of sole leather. This is repeated as indicated; usually only one treatment is given and the patient is advised to come in a month after delivery for the operation.

The operation done in severe cases must be radical. Recurrences and, what is more common, continuation of symptoms with only partial relief and which later become severe, are more common with incomplete operations upon this group than in the mild goiters. The entire thyroid gland is diseased in all except the isolated adenoma. The types I have been discussing are usually caused by the diffuse types of goiter. The patients belong to the desperate goiter group because of the severity of reaction to the secretion of a pathologic gland. A partial operation that leaves sufficient pathological gland to continue to produce the secretion does not cure. The operation must be so radical that were regeneration not to take place, myxedema must result because of a lack of secretion.

In experimental work I have demonstrated that regeneration with the development of normal thyroid tissue sufficient to meet the needs of the experimental

animal will take place if the gland is saturated with iodine and kept saturated for two months after the operation. In practice, I give 10 minims of Lugol's solution three times a day for one month after the operation and then once a day for the second month, and follow this with iodized salt or 10 mg. of iodine weekly in tablet form. I have never seen a recurrence when this method has been followed. Sometimes there will be a transitory hypothyroidism as a result of the radical operation but this is easily controlled with desiccated thyroid in small doses and has always cleared up, in patients I have seen, within one to three months.

Experimentally we have demonstrated that without the pre- and postoperative saturation of the thyroid with iodine, excessive hyperplasia with true goiter development may occur.

In conclusion I wish to state that in my judgment, with careful study and careful handling before the operation, the desperate goiter patient does not offer a much greater risk than the milder case so far as the cure of the goiter itself is concerned. However, permanent lesions existing before the operation will still be present so that many patients will still have some of the effects of the severe goiter although the toxic goiter has been replaced by a thyroid gland with a perfectly normal secretion.

THE RELATION OF GOITER TO OTHER ENDOCRINE GLANDS*

JAMES H. HUTTON, M.D.

CHICAGO

NATURALLY only the high points in such a discussion can be touched upon in a paper of this kind. Let me say in the beginning that there is considerable doubt in my mind as to whether exophthalmic goiter is a disease of the thyroid. A few reasons for this attitude are as follows: We cannot accurately correlate thyroid pathology and goiter symptomatology, that is, the clinician cannot foretell from the symptoms and the preoperative physical and laboratory findings what the pathologist will find when the gland is removed. And the pathologist cannot tell from the section what symptoms the patient may have exhibited. Most of the symptoms of Graves' disease may be present without goiter. The fact that many patients are relieved of most of their symptoms by removing most of their thyroid tissue does not prove that the thyroid is the only, or even the chief, factor at fault in this syndrome. This post hoc, ergo propter hoc line of reasoning has very little standing in the scientific world outside this particular field. Our ignorance of the etiology and pathogenesis of Graves' disease is almost as complete now as it was two decades ago.

Perhaps the easiest way to go about such a discussion as this will be to take up the various glands most intimately related to the thyroid and to try to find out how much we know about this relationship, how it affects the etiology, symptomatology, prognosis and treatment of goiter, and what we can do about it, if anything.

The pituitary, the parathyroids, thymus, adrenals, gonads, and the pancreas are the glands most intimately related to the thyroid.

THE PITUITARY

Embryologically the relationship between the anterior lobe and the thyroid is very close. This relationship is probably continued in a functional way throughout life. The anterior lobe is formed from a mass of epithelium split off from the pharynx just as is the thyroid. But in the male a larger mass goes to the pituitary than goes to form the thyroid, while in woman the reverse is true. Is this of any significance? The majority of toxic goiters, especially the exophthalmic variety, occur in women. Eason[1] says most of these occur in women of narrow frame, of light skeletal structure with associated narrow subcostal angle and enteroptosis, individuals whose structure would make one think perhaps the anterior lobe of their pituitary had not been sufficiently active during their prepubertal years. It rarely occurs in men of strong rugged frame, showing evidence of an anterior lobe very active during the developmental years.

In acromegaly, which is presumably due to hyperfunction of the anterior lobe, the thyroid, parathyroids, adrenal cortex, and gonads show hyperplasia. Presumably, the opposite is true. That is, in Simmonds' disease the same structures show some degree of atrophy. Cushing[2] says in states of pituitary deficiency these particular glands are found to be atrophic.

THE PARATHYROIDS

The parathyroids are of great interest to the thyroid surgeon because of their intimate anatomic relation to the thyroid. It is now conceded by every one that the parathyroids are not derived from the thyroid. A good many patients following

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thyroidectomy show some signs of parathyroid insufficiency. These symptoms are not usually due to the removal of the parathyroids, but because their blood or nerve supply has been interfered with, either at the time of operation or by scar tissue formation. It is important that such cases be promptly recognized and properly treated. The gland may be sufficiently active to perform its function during ordinary strains and only show signs of insufficiency during unusual stress as during the menstrual periods or pregnancy or when the patient becomes constipated. It is probable that the parathyroids bear somewhat the same relation to calcium metabolism that insulin does to carbohydrate metabolism and that in the presence of a severe degree of parathyroid lack, the body is unable to utilize calcium even when it is present in sufficient quantities. In such cases, it is necessary to use the parathyroid hormone for a time. The body eventually accustoms itself to the loss of much, if not all, of its parathyroid tissue. During this period of treatment or readjustment the patient should be given considerable doses of calcium and should be kept on a low proteid diet. The use of citrus fruits is helpful. Calcium should be given in large doses. It is most conveniently given as the lactate in doses of 1 dram t.i.d. in a glass of water with lactose drams 3. Luminol in doses of 1 to 1½ grains t.i.d. is helpful in allaying the nervous disturbances. The calcium is many times more effective if given intravenously, using a solution of the chloride or glucocalcium. This method can usually be stopped after a few days.

THE THYMUS

For a long time pathologists have known that thymus tissue existed in some quantities in toxic goiters, especially the exophthalmic variety. Hypertrophy of the thymus and hyperplasia of the lymphoid tissue generally are characteristic of Graves' disease. The lymphocytes show a relative and absolute increase in exophthalmic goiter. It has been suggested that in cases showing a hyperlymphocytosis, operation is a dangerous procedure. Consequently a white and differential cell count should be made before operation. Careful physical and roentgen-ray examination should be made to discover if an enlarged thymus is present. If found, it should be properly radiated before thyroidectomy is done. It is not beyond the range of probabilities that cases showing the greatest improvement under roentgen-ray therapy are those having considerable amounts of thymus tissue. The fact that such enlarged glands have not been noted does not rule out the likelihood of their presence.

THE ADRENALS

Adrenaline heightens the effect of sympathetic impulses and exaggerates the symptoms of Graves' disease. On this basis Goetsch built his test for hyperthyroidism. This is probably of greater negative than positive value. While many persons respond positively to it who do not have Graves' disease, cases that do not respond to it are undoubtedly not cases of Graves' disease. The administration of adrenaline by mouth to a patient having Graves' disease will usually produce a gastrointestinal upset. The more severe the symptoms of Graves' disease, the more easily is this upset produced.

The Adrenal Cortex. The only time exophthalmic goiter has been produced experimentally was by damaging the adrenal cortex. Using rabbits, because they are the only experimental animals having sufficient accessory adrenal tissue to withstand double adrenalectomy, Marine and Baumann[3] were able by removing both adrenals and so producing sublethal damage to the adrenal cortex, to reproduce all the symptoms of exophthalmic goiter, including exophthalmos and goiter. There was tremor, tachycardia, weakness, loss of weight and strength, increased nervous instability, increased heat production, hypertrophy of the thymus, hyperplasia

of the lymphoid tissue and increased sensitiveness to toxins.

Following this work, Shapiro[4] used an emulsion of fresh adrenal cortex in glycerin in the treatment of Graves' disease. He regards it as a valuable remedy though by no means a cure-all. It is possible that we shall find eventually that many of the symptoms of Graves' disease are due to insufficiency of the adrenal cortex. One sees some cases exhibiting the usual symptoms of Graves' disease but having neither goiter nor an elevation of the basal metabolic rate. It is possible that these are cases of adrenal cortex insufficiency. Treatment along the line suggested by such an idea produces very satisfactory results, whereas if these cases are allowed to proceed untreated, they finally develop the typical picture of Graves' disease. The' number of such cases in the literature is increasing.

THE PANCREAS

There are certain striking resemblances between diabetes mellitus and Graves' disease;[5] liver glycogen is deficient in both, tissue storage of carbohydrate is deficient in both, nitrogen excretion is excessive in both, blood sugar is excessive in both, carbohydrate tolerance is reduced in both, glycosuria is fairly constant in diabetes and occasional in Graves' disease, emaciation is constant in both, the appetite is good, even excessive in both, the conversion of sugar to glycogen is defective in both, the respiratory quotient is low in both.

Insulin is of some value in the treatment of Graves'; it tends to keep the temperature near normal. By giving relatively small doses with glucose, the advantages to be derived may be summed up as follows:

1. The intermittent loss of sugar is prevented.

2. More sugar is available for withdrawal to the tissues for storage.

3. The excessive combustion of sugar, due to the action of thyroxin and adrenaline, is restricted, an adequate store of glycogen is maintained in the liver.

4. The tissue starvation of carbohydrate and phosphorus and possibly of amino acids is diminished.

5. The function of the liver in forming glycogen from fat and proteid under the stimulus of thyroxin is controlled.

6. The protein and fat-sparing function of insulin diminishes ketosis and possibly diminishes the amount of fat in the liver.

There are no special risks in using insulin in the treatment of Graves' disease. The body shows a decreasing tolerance for it so that the dose has to be cut down after a few weeks or the amount of glucose given with it needs to be increased. The latter is the better procedure as by this the weight of the patient may be maintained or even caused to increase.

THE GONADS

There has been much discussion among endocrinologists as to whether the thyroid and the gonads were synergists or antagonists. In the present state of our knowledge it is impossible to settle this question. The following facts are significant. The thyroid has a stimulating while the gonads have an inhibiting effect on the growth of long bones. Thyroid insufficiency is accompanied by a considerable tendency to obesity. This is absent or much less marked in primary hypogonadism.

Graves' disease is much more common among women than among men. The ratio being variously estimated at from 5:1 up to 10:1. This is during her most active sexual life. After the menopause the ratio sinks to 3:1. More cases of myxedema in women occur after the menopause than at any other time.

The intimate relation of the thyroid and the ovaries is suggested by the term "uterus of the neck" which is sometimes applied to the thyroid. Colloid goiters make their appearance at puberty. The thyroid enlarges during the menstural periods and pregnancy. During pregnancy the B.M.R. is usually elevated 10 to 20

per cent. Women having adenomas without hyperthyroidism frequently feel better during pregnancy than at any other time. On the other hand adenoma without hyperthyroidism that may have been present for many years becomes active at or near the menopause.

Enlargement of the thyroid occurs in animals from which the ovaries have been removed. Eason[1,6] says the female is predisposed to thyroid disorders. But hyperthyroidism seldom occurs in either sex until the adult development of the genital and interstitial cells has been attained. At puberty the thyroid becomes larger and more vascular, contains more iodine and shows evidence of greatly increased activity. Such signs are much more common in the female; while in the male the neighboring organ, the larynx becomes enlarged.

Quoting Eason[7] again, he says:

> At this stage, i.e., puberty, the growth of the thyroid gland seems to be stimulated by the endosecretions of other glands. Of these the interstitial cells of the ovary and testis are the most important (by their action either directly on the thyroid or indirectly by nervous and mental processes).

Many simple goiters show signs of activity at this time and there is little question but that many are needlessly removed. There are some signs of hyperthyroidism associated with these enlargements, but the basal metabolic rate is usually near normal. The proper exhibition of some form of ovarian medication seems to produce satisfactory relief of these symptoms.

Severe degrees of hyperthyroidism are usually accompanied by considerable disturbances of the menstrual function or even by amenorrhea.

The menstrual history of a woman having hypothyroidism is characterized by the early onset of the menstrual periods, their regularity, profuseness, and freedom from pain. She usually begins to menstruate at an earlier age and continues longer than the woman having signs of hyperthyroidism.

Finally, in examining goiter patients let us not focus our attention entirely on the thyroid or on symptoms that in our present knowledge are believed to arise as a result of its dysfunction. Let us not forget that the patient has other endocrine glands besides the thyroid and that some of these may not be functioning properly. The recognition and proper treatment of these disorders will at least not delay the patient's recovery.

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INJURIES TO THE PARATHYROIDS
& SUBSEQUENT MANAGEMENT*

CONSTANTINE F. KEMPER, M.D.

DENVER

A COMPREHENSIVE discussion of the parathyroid glands, calcium metabolism and tetany is beyond the scope of this paper. The voluminous literature embracing the development of our knowledge of these related subjects has been exhaustively reviewed and is easily available. Conspicuous among these is a monograph by Boothby which was published in 1922. Since that time the story of the epoch-making work of Collip, in his isolation of the calcium-raising hormone, has been added to the literature. Very recently Dragstedt has reviewed the conflicting theories of the function of the parathyroid glands and summarized the evidence for and against each of these in the light of his extensive animal experimentation. From these sources, with their complete bibliographies, reliable references may be had to the best that has been thought and the most that is known concerning the parathyroid glands. In this discussion the subject is necessarily limited to the concrete problem of accidental injuries occurring at time of operation with specific emphasis on the treatment of their sequelae.

One of the serious complications of thyroid surgery is some type of injury to the parathyroid glands. Damage to these important adjacent structures may consist of their complete removal along with the thyroid. It has also been reported that they are sometimes injured by the grasp of a hemostat or by some direct interference with their blood supply. From the gradual and delayed onset of symptoms it is believed that they and their blood supply are rarely injured by the encroachment of scar tissues. Whatever the nature of the injury, it seems well established that when two or more of these glands are rendered functionless, parathyroid tetany occurs.

Fortunately, improvement in surgical technique and a more general appreciation of the possibility and seriousness of such an accident have reduced the percentage incidence of parathyroid tetany following thyroidectomy; but the enormous increase in the total number of thyroidectomies performed, the recognized necessity for secondary operations in recurring types of toxic goiter and the occasional indication for radical surgery at any cost continue to produce more than a negligible number. While complete and authentic statistics of its incidence are not available, surgeons doing an enormous amount of goiter surgery have reported that parathyroid tetany occurs following $\frac{1}{10}$ to $\frac{1}{2}$ of 1 per cent of the thyroidectomies done. It is probable that this difference of incidence is more dependent upon the failure of some surgeons to report cases of mild and transient tetany than upon differing operative procedures. But mild or severe, transient or permanent, it still occurs often enough to demand further prophylactic efforts.

The problem of prevention is purely a surgical one, which can well be left to the surgeon. It is questionable, however, whether the percentage incidence of parathyroid injury can be expected to be appreciably reduced by further surgical caution based on the conventional understanding of the anatomy of these structures. It is well to recall that the parathyroid bodies are relatively small glands firmly attached to the thyroid capsule. While they have characteristics which some anatomists contend make them

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"easily recognizable" this is not true under the well known conditions that prevail during an operation. While it is known that, as a rule, the two pairs of glands have a characteristic relation to the thyroid and its arteries, anomalous positions do occur. Besides, these tiny structures share with the thyroid a common blood supply. Under these anatomical conditions and with the occasional necessity for radical and secondary operations, the wonder is not that they are sometimes injured, but that they are almost always spared.

For many years surgeons have advocated reimplantation of parathyroid tissue, if, during the course of an operation, it is discovered that such tissue has accidentally been removed. Millzner, Terry, and their associates have recently demonstrated that anomalous parathyroid tissue may frequently be found on the anterior capsule which, they believe, if spared by the surgeon, may hypertrophy and take on compensatory function. They have also shown that by an ingenious technique this tissue may be identified in the removed thyroid and successfully reimplanted. Lahey working independently has likewise advocated a similar procedure at the operating table. It is too early to conclude the extent of its practical value but by some such procedure it is reasonable to hope for a reduction in postoperative tetany that would otherwise occur from unavoidable injury. The grafting of parathyroid tissue from other individuals or lower animals has not yet been justified by experience. The inherent difficulties encountered in heteroplastic and zooplastic grafting of other organs does not offer much encouragement when applied to the parathyroid glands. With the exception of reimplantation of identified tissue at the time of operation, successful gland grafting is still a far cry. Consequently tetania parathyreopriva still occurs giving rise to a rare but extremely difficult problem of medical management. It is the chief purpose of this paper to consider the therapeutic means now in use and to report the results of their use in the treatment of 2 patients of chronic and severe parathyroid tetany.

It is well known that this condition occurs in an acute and a chronic form. Much confusion exists in the literature in evaluating methods of treatment because of failure to separate these types. The acute or transient form comes on very soon after operation and disappears in from one to ten days with or without treatment. The parathyroid glands undoubtedly share in the postoperative edema and irritation and for this reason are probably temporarily deprived of their blood supply. This hypothesis for the cause of temporary hypofunction seems wholly tenable when it is recalled how often demonstrable swelling and irritation of the trachea follow thyroidectomy.

Treatment in such cases is very simple. In acute tetany 20 c.c. of 5 per cent calcium chloride solution is administered intravenously whenever the muscular contractions are painful and repeated when they recur. The patient gets well not because calcium or any other drug has been given but because edema of the neck structures subsides, circulation is restored, and the parathyroid glands take up their normal function. Such patients because of their transient tetany and tendency to recover without treatment cannot be used as test cases of therapeutic agents save for the demonstration of the immediate and spectacular value of intravenous calcium.

Chronic tetany is an entirely different problem. Here the parathyroid bodies or their blood supply are destroyed beyond restoration. Save for the chance of developing compensatory function of parathyroid tissue ordinarily inadequate, such a patient must come to depend upon some type of more or less regular medication indefinitely. Despite the unexplained tendency of chronic tetany to periods of activity and remissions it is this type which offers the most crucial test of treatment methods.

In 1909, MacCallum and Voegtlin demonstrated that hypocalcemia is asso-

ciated with parathyroid tetany. They further demonstrated that the injection of calcium into the blood stream in sufficient amounts immediately relieves the test animal of its tetanic spasms. They therefore concluded that the parathyroid glands in some way control the calcium metabolism, thus maintaining a constant blood level of about 10 mg. per 100 c.c. While it is admitted that there may be other functions of the parathyroids, their significance in the control of blood calcium has not been seriously questioned. Since that time calcium has been the sheet anchor in treatment. But it was soon observed that calcium salts, when given by mouth, had little to do with the blood calcium level. Diets rich in calcium and the drug in enormous amounts given preferably when the stomach is empty have been advocated but it has not been conclusively shown that any such regime raises the blood calcium appreciably in the absence of adequate parathyroid secretion. While the drug is highly successful in temporarily relieving symptoms and raising the blood calcium when given intravenously, it is *practical* only in *acute* tetany. To attempt to treat chronic tetany by such a method would demand the daily attention of a physician. It would likewise subject the patient to the constant danger of a slough at the site of injection, scars from which are the telltale marks of unwise therapy in many cases of tetany. Calcium *alone* is impractical or ineffective.

Evidence of specific effects in the substitution method of feeding desiccated parathyroid has not been convincing. Collip has more recently shown that the active extract is rendered inert by the digestive ferments. Therefore, unless there is demonstrated some active principle other than the so-called hormone of Collip, gland feeding is useless. Conflicting and inconclusive clinical evidence would also seem to substantiate this belief. A practical method of substituting for the loss of parathyroid activity, then, must be something other than gland feeding.

In 1924, Collip isolated an active extract or hormone which when given intravenously or subcutaneously causes a rise in the blood calcium of experimental animals. These definite findings are too recent and too well confirmed by others to require further comment. The hormone has been standardized in terms of units, 1 unit being one $\frac{1}{100}$ of that amount which will raise the blood serum calcium of a 20 kilo dog 5 mg. in fifteen hours. If this hormone, so active in some lower animals, is commensurately accurate in man, it would seem perfectly obvious that chronic tetany could be controlled by hormone injections much as diabetes mellitus is controlled by insulin therapy. It would seem that all we need to do is to provide an excess of calcium in the dietary and administer adequate amounts of parathyroid hormone to maintain the blood calcium between 8 and 12 mg. This adequate dosage of hormone could apparently be ascertained by blood calcium determinations much as the insulin dosage is worked out against glycosuria and blood sugar. In 1926 Collip reported 1 such case so standardized. He further reported gratifying clinical results in the hands of Crile, Lisser and others. More recently John has reported that the administration of parathormone is followed by relief in all cases of chronic tetany. To these clinical experiences I wish to add the following typical cases:

CASE REPORTS

Case 1. A. C., female, aged forty-six, was admitted to the Colorado General Hospital March 20, 1928. She had been treated for parathyroid tetany for five years. The significant part of her history is as follows: Developed exophthalmic goiter for which she had a thyroidectomy in 1916. Recovery uneventful and enjoyed fair health up to July, 1922, when goiter symptoms returned. Hospitalized for three weeks and received two radium irradiations of the thyroid. Had a ligation followed by a thyroidectomy in January, 1923. Says she began to have muscular pains and convulsions before she returned from the hospital for which she received calcium intravenously. Despite calcium and desiccated parathyroid by mouth she continued to have one or two convulsions

Chart 1, Case 1

CHRONIC PARATHYROID TETANY

Date 1928	Parahormone	Calcium Lactate Grs.	Blood Chemistry			Notes
			Calcium	Phosphorous	Plasma CO_2 Combining Power	
Mar. 20	..	750	...	...	....	Had several attacks of pain. "Hysterical spells."
21	..	750	9.6	...	....	Same as previous day.
22	..	750	...	...	....	One attack in twenty-four hours. More cheerful.
23	..	750	...	...	....	Restless. Feared attacks but had none.
24	..	750	...	...	64.2	Quiet, not nearly so nervous.
25	30	300	...	...	....	Definite convulsion 7:00 A. M. Dazed after the attack.
26	30	300	...	...	....	Fair day.
27	30	300	...	...	....	A much better day. No complaint.
28	30	300	7	6.9	....	A better day. Much improved. No complaint.
29	30	300	...	...	....	Patient happy. No complaint.
30	30	300	...	...	....	Nauseated. No complaint.
31	30	300	...	...	....	Good day. Out of bed.
Apr. 1	30	300	...	...	....	Good day.
2	30	300	...	...	....	Good day.
3	30	300	...	...	....	Good day.
4	30	300	9.2	...	....	No complaint.
20	30	300	8.7	...	....	No complaint.
26	30	300	7.2	...	....	No complaint.
May 22	15	300	9.7	...	....	No complaint.

daily. During next two years frequency of the convulsions became less.

In 1925 convulsions became severe and frequent just preceding appendectomy, but subsided after operation. During the last three years her condition has shown marked improvement. Has noticed she gains weight and is free from attacks during summer months. Begins to go down in winter, loses appetite and weight and has considerable muscular pain. Convulsions become more frequent reaching their maximum about March, to subside again with the coming of summer. At the height of seasonal tetany she was admitted to the Colorado General Hospital, March 20, 1928, for study and treatment. Her subsequent history is set forth in Chart 1.

Case II. W. H. D., male, aged thirty-eight, seen September 23, 1927. His complaint and physical findings were typical of goiter of the exophthalmic type. His family and past history were not significant. Present complaint began in 1922. First thyroid surgery was in 1925: one operation in July, another in August and a third in September of that year. His neck shows two lateral and one collar scar. Felt better for almost one year after these operations but the same type of symptoms returned in 1926. Since he received little or no relief from medical treatment he was again operated on October 14, 1927. He made an uneventful recovery save for some tingling in the skin and extremities. This new complaint persisted and by the middle of November, 1927, he was having considerable pain especially in the extremities and chest, also typical carpopedal spasms. He was given calcium and desiccated parathyroid gland by mouth without relief. Twenty c.c. 5 per cent calcium chloride gave immediate relief which lasted for three days when it was repeated with relief again. He then improved in every way for about one month, at the end of which period muscular spasms and pains returned and were uninfluenced by 20 units of parahormone plus large doses of calcium by mouth, but were relieved by intravenous calcium. His spasms showed no signs of

CHART II A, CASE II
CHRONIC PARATHYROID TETANY

Date	Parahormone	Calcium Lactate	Blood Chemistry			Clinical Notes
			Calcium	Phosphorous	Plasma CO_2	
1928 Jan. 20	30	...	....	.....	.....	Very restless.
21	30	...	6.6	.....	.....	Feels fairly well.
22	..	...	....	.....	.....	Pain in the neck, chest, back and arms.
23	..	...	....	.....	.....	Pain in arms and hands.
24	..	...	6.2	.....	.....	Pain in arms, kept to bed.
25	..	...	....	.....	.....	Pain in hands, blurring of vision.
26	..	...	....	.....	....	Choking spells.
27	..	...	5.8	.....	.....	Choking spell. Convulsion unconscious for a few minutes. 20 c.c. $CaCl_2$ 5 per cent intravenously.
28	60	...	5.4	.....	.....	Complains of sore mouth only.
29	60	...	....	.....	.....	No complaint.
30	60	...	....	.....	.....	No complaint.
31	60	...	....	.....	.....	No complaint.
Feb. 1	60	...	5.9	.....	.....	No complaint.
2	60	...	5.15	.....	.....	Complained of pains in hands, arms and no sleep. 2 gm. $CaCl_2$ intravenously.
3	60	...	5.3	.....	.....	Marked pains, codeine grain ½.
4	60	...	....	.....	.....	Severe pain, unable to sleep, codeine grains 2½.
5	25	300	....	.....	.....	Pains; .5 gm. $CaCl_2$ intravenously.
6	25	300	....	.....	.....	No complaint. First day of meat-free diet.
7	25	300	6.91	.....	.....	No complaint. Meat-free diet.
8	25	300	....	.....	.....	No complaint. Meat-free diet.
9	25	300	....	.....	.....	No complaint. Meat-free diet.
10	25	300	....	.....	.....	No complaint. Meat-free diet.
11	25	300	7.9	.....	.....	No complaint. Meat-free diet.
12	25	300	....	.....	.....	No complaint. Meat-free diet.
13	25	300	....	.....	.....	No complaint. Meat-free diet.
14	25	300	7.2	.....	.....	No complaint. Meat-free diet.
15	25	300	....	.....	.....	No complaint. Meat-free diet. Patient went home, apparently normal.

remission, so he was sent to the Colorado General Hospital February 10, 1928. Since that time he has been on a meat-free diet with calcium chloride grains 100 and cod-liver oil, three times a day. Chart IIB shows what if any effects the hormone had on his blood calcium and symptoms.

SUMMARY

1. Further surgical prevention of parathyroid tetany seems to depend upon the development of the technique of reimplantation of parathyroid tissue at time of operation, especially in patients operated two or more times.

2. Calcium by mouth is of little value in the absence of adequate parathyroid tissue. It is impractical to give it intravenously in chronic tetany.

3. The feeding of dessicated parathyroid gland seems useless.

4. The subcutaneous administration of the parathyroid hormone in dosages as usually given has little, if any, influence on the blood calcium level. Apparently it should be given in much larger dosages.

5. The clinical symptoms seem to be as good a guide to the amount given as the blood calcium level.

CHART IIB, CASE II
HOME ON MEAT-FREE DIET FOR ABOUT TWO AND ONE-HALF MONTHS

Date	Parahormone	Calcium Lactate	Blood Chemistry			Clinical Notes
			Calcium	Phosphorous	Plasma CO_2	
1928 Feb. 18	30	300	7.0	...	..	Doing well.
24	30	300	7.0	...	..	Doing well.
Mar. 5	30	300	7.2	...	..	Doing well.
14	30	300	6.4	...	..	Doing well.
20	30	300	7.0	4.9	63	Doing well.
29	30	300	9.0	...	..	Doing well.
Apr. 3	30	300	8.3	...	..	Doing well.
20	..	300	8.1	...	..	Doing well.
26	..	300	8.6	...	..	Doing well.
27	..	300	...	...	..	Lost appetite. Pain in stomach, complained of head cold. Slight constriction in chest.
29	40	300	4.3	...	..	Definite tetany symptoms. Unable to secure weekly blood calcium, was necessary to give 1 or 2 gm. calcium chloride intravenously daily until he was readmitted to hospital.

CHART IIC, CASE II
REENTERED HOSPITAL MAY 28, 1928

Date	Parahormone	Calcium Lactate	Blood Chemistry			Clinical Notes
			Calcium	Phosphorous	Plamsa CO_2	
1928 June 2	60	300	4.7	5.2	.....	Very restless. Luminol grains 6. $CaCl_2$ 2 gm.
3	90	300	...	...	.....	Luminal grains 4½. Patient very restless.
4	90	300	...	...	.....	Luminal grains 4½. $CaCl_2$ 2 gm. Stuporous.
5	110	300	...	...	.....	Luminal grains 7½. Very restless.
6	120	300	...	...	.....	Luminal grains 6. Very restless.
7	120	300	4.9	...	.....	Luminal grains 4½. Slightly improved.
8	120	300	...	...	.....	Luminal grains 4½. Codeine grains ½. Not so restless. Pains in arms, severe.
9	120	300	2.5	...	.....	Luminal grains 4½. $CaCl_2$ 1 gm. Patient restless and emotional. Severe pain, seems irrational at times.
10	120	300	...	...	.....	Luminal grains 6. Says he does not feel well but no definite pain.
11	300	300	5.5	...	.....	Luminal grains 4½. $CaCl_2$ 1 gm. Very restless and emotional but became quite normal after $CaCl_2$.
12	50	300	4.0	...	.....	Luminal grains 4½. Complains of itching sensation but no pain. Not emotional. Up and about and for the first day since admission seems nearly normal.
13	50	300	...	...	.....	Luminal grains 4½. Comfortable.
14	50	300	7.1	...	.....	Luminal grains 4½. Comfortable.

AN INTERPRETATION OF THE CLINICAL HISTORIES OF MORE THAN 1000 TOXIC GOITER PATIENTS*

B. T. KING, M.D.

SEATTLE

THE material contained in this paper is drawn from the clinical histories of more than 1000 patients who have been given a diagnosis of toxic goiter. More than 800 have been operated upon and the diagnosis confirmed by pathological section and the clinical cure or improvement of the patient. No patient has complained of all the symptoms mentioned and the symptoms have varied greatly in different individuals, due chiefly to age, duration and severity of disease, and type of goiter, whether exophthalmic or adenomatous, though no effort will be made in this discussion to differentiate between the histories of the two types of toxic goiter. A majority of the patients presented themselves without previous examination for their complaint or had been referred with correct diagnosis already made.

Previous erroneous diagnoses have occurred with sufficient frequency to warrant mention of the diseases for which toxic goiter has been mistaken, and these will be mentioned in the order of frequency in which they occurred. It is fair, I believe, to assume that such diagnoses will be made with less frequency in a community where toxic goiter is infrequently encountered. A number of these patients have consulted from one to ten different doctors. Too often this has served to add a confusion of diagnoses to an already bewildered patient. It therefore seems that a review of the complaints of these people may be of value.

The diseases for which toxic goiter has been mistaken will be mentioned first so that they may be borne in mind in a discussion of the symptoms. No attempt will be made to give a complete historical differentiation between these diseases and toxic goiter.

The diagnosis "nervous breakdown," ambiguous term at best, stands distinctly at the head of the list of mistakes. It occurred perhaps as frequently as all others combined. Next is heart disease; 3rd, influenza; 4th, intestinal flu; 5th, nervous indigestion; 6th, hypertension; 7th, various psychoses, nephritis, tuberculosis, gynecological disturbances, etc. "General run-down condition" has done overtime as a diagnosis. One man had his gall bladder removed for the jaundice of an exophthalmic goiter. Numerous others had had teeth and tonsils removed. A few appendices had been removed in cases with pronounced gastrointestinal symptoms.

Almost without exception the patients have been made worse by any operative procedure. I have seen a number of patients in whom a crisis had been precipitated by the removal of tonsils or the extraction of teeth. The patient with toxic goiter, be it exophthalmic or adenomatous, is poorly equipped to withstand any sort of trauma. I mention this because of a pernicious idea that still prevails regarding the treatment of the goiter patient.

A few years ago a number of articles appeared in medical literature relative to the benefits of tonsillectomy to the goiter patient. These were chiefly concerned with associated goiter of adolescence. Others attempted to hitch up focal infections and exophthalmic goiter. As I have seen it, the unhitching has proved rather sad experience for the toxic goiter patients. If focal infection of a damaging nature be pres-

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ent, it should be left until the goiter is removed. The foregoing list of mistaken diagnoses, it will be noted, includes diseases referable to almost every organ in the body. But as toxic goiter is essentially a disturbance of so vital a function as metabolism, there is no organ in the body that may not be affected by a disturbance of this function.

The symptoms complained of by a given patient as a rule have been multiple. A majority have experienced all three classical symptoms of the disease, namely, nervousness, tachycardia and loss of weight. In other patients there has been a tendency for one system or group of organs to be affected more or less than the others. In some, nervous symptoms have predominated; in others, cardiovascular; in others, gastrointestinal; in others, muscular symptoms. It is likely that these variations are more dependent upon individual susceptibilities or constitutional inherited tendencies than upon any selective action of the thyroid secretion for any particular tissue. In the aggregate, symptoms have been encountered referable to every tissue and organ in the body, with the possible exception of the bones.

HEART SYMPTOMS

The most frequent single complaint is referable to the heart, yet not infrequently one encounters a patient with toxic goiter who gives few or no symptoms referable to it. The symptom that occurs with the greatest frequency is tachycardia. Patients say that the heart beats hard and fast or pounds. The average patient is more often conscious of this pounding when lying down than in the upright position. Few symptoms occur with more frequency or are more significant from a diagnostic standpoint than inability of the patient to sleep, or at least sleep comfortably, on the left side. In this position the apex of the heart rests against the rigid chest wall and pounding there becomes annoying and disturbing. In the upright position, the heart is suspended and the apex does not strike the chest wall with such force. When the patient is on the back or the right side, the semi-elastic pericardial sac absorbs much of the shock of the apex beats. In conjunction with the foregoing it has been an interesting observation that the pain complained of in the region of the heart by the goiter patient is more often at the apex than at the base, such as occurs in anginal and allied conditions. Several patients have complained of a tenderness of the left breast; others of sensitiveness of the skin in the region of the apex, due no doubt to the effect of the pounding ventricles on the intercostal nerves. In some patients the heart beats hard and fast continuously. In others the attacks come on without apparent cause. In nearly all the cardiac reaction to exercise and excitement is out of proportion to the degree of effort or the stress of circumstances.

By questions any history of irregularity of heart action and edema of the ankles, should be elicited as these symptoms give evidence of cardiac damage and decompensation. Furthermore the history of rapid and irregular heart action becomes a diagnostic clue of the greatest importance. Particularly is that true in communities where goiter is endemic. I once heard Plummer of Rochester state that 90 per cent of the cases of auricular fibrillation that occurred in the Mississippi valley were due to goiter. In my own experience the percentage has been greater than 90 per cent. If one can diagnose the cause of auricular fibrillation in 90 per cent of the cases, it is a very high average and toxic goiter should always be considered when one finds a rapid irregular heart. Occasional extrasystoles are less significant. Relative to increased pulsations, in extreme cases the patients may feel a pulsation throughout the entire body, but the majority of them will only have noticed it in the neck and the epigastric region, due respectively to increased pulsations in the carotids and the abdominal aorta. The range of disturbed cardiac activity is

extremely wide and varies from the violent heart beats that shake the bed on which the patient lies or are audible for a few feet down to slight attacks of palpitation or shortness of breath on exertion.

GASTROINTESTINAL SYMPTOMS

A surprising number of patients have centered their complaints on some gastrointestinal disturbance. The story generally runs something like this: "A few months ago I began to get nervous and shaky and lost my appetite. I was nauseated for a few days or a few weeks at most. Any kind of food caused distress: a sensation of fulness in the upper abdomen. This passed off and then I had an abnormal appetite, could eat anything without distress, was always hungry, but continued going down and losing weight and strength. About the same time, I noticed my heart beating hard and fast. I was warm all the time and perspired freely." Others have had a painless and unexplainable diarrhea; some have vomiting and this, unless of short duration, becomes a very serious phase of the disease. There is no other disease that gives the foregoing history. A diagnosis can be established with practical certainty on such a history.

I have seen a number of patients whose chief complaint was nausea and vomiting accompanied by a very distressing pulsation in the epigastrium. Several such had been previously given a diagnosis of aneurysm of the abdominal aorta. I know of no disease except exophthalmic goiter that causes nausea and vomiting accompanied by pronounced pulsation in the epigastrium. Without exception, these symptoms have been relieved by the removal of the goiter.

It is difficult on a physiological basis to explain the symptoms of nausea vomiting, and diarrhea, unless they are of a toxic nature. It is in this group of patients that the erroneous gastrointestinal diagnoses have occurred. I always suspect toxic goiter in any adult who gives a history of having had "intestinal flu" or painless diarrhea. Gastrointestinal disturbances in toxic adenoma are not frequent, but occur occasionally in the more active phases of the disease.

NERVOUS MANIFESTATIONS

The nervous symptoms have been many and varied, the more frequent having been described as a "feeling of nervousness." The patients are restless, impatient, irritable and cranky. They lose the power of concentration and continued effort. Small annoyances disturb them out of proportion to their importance. Increased excitability is a most constant symptom, accompanied by an increased physical response to excitement and emotional disturbances. They often say they become so weak and shaky from excitement or from being startled that they are almost helpless. The ringing of the telephone or door bell may upset them markedly. Emotional disturbances are frequent. They cry or get angry about nothing at all. They are difficult to manage both at home and abroad. In contradistinction to the neurasthenic, the goiter patient is a hopeful or optimistic individual. He believes in himself and his ultimate recovery. He may and often does have periods of depression. He may lose interest in his work but not in himself.

Of all the nervous symptoms the one that most concerns the patient is tremor. To her it is frequently the most conspicuous symptom. However, it is not purely a nervous but a neuromuscular symptom. She notices it when writing, eating, threading a needle, etc. She spills coffee or tea over herself or her friends, drops dishes and does various other embarrassing things. Women often go so far as to avoid social functions, teas, etc., for this reason, and because they do not wish other women to think they are easily fussed or confused. In addition to the tremor of the hands and knees they often describe a trembling, shaky feeling inside them. They cannot locate it, but when they have this feeling they usually also have an exophthalmic goiter.

MENTAL SYMPTOMS

A true psychosis has been encountered only once in this group of cases. The patient had been confined in an insane asylum for violent outbursts of temper and because she sat out in the rain clothed only in her nightgown. These outbursts could be explained by the presence of an exophthalmic goiter. It is also possible that she sought relief from an excessive heat production by recourse to the only available shower, as at the time of the onset of her illness she resided in the country, where bath tubs and showers were non-existent. It is now more than two years since she was operated upon and while at present she is no mental giant, her condition is in no way different from her previous normal mental and physical state.

Several cases of delirium and coma were encountered. These are as a rule of short duration, chiefly because patients die in this condition, unless it is checked in a short time. However, one patient was delirious intermittently for nearly three months.

NERVOUS BREAKDOWNS

It has been an interesting observation that from 20 to 30 per cent of all toxic goiter patients give a history of one or more nervous breakdowns, also that fully 90 per cent of those who have given such histories on careful analysis, have been found to describe clinical symptoms strikingly parallel to exophthalmic goiter, and should have been so diagnosed. Nervous breakdown is a dangerous diagnosis and will lower your batting average if you stick to it. The history of a nervous breakdown is a most important clue and generally narrows a diagnosis down to one of two groups of condition, namely, the neurasthenic group, including the nervous exhaustion and neurocirculatory asthenic types and the toxic goiter states. If one will think of a nervous breakdown as in the majority of instances signifying one of the two conditions, it will help to clarify many diagnoses. It might be said that a basal metabolic determination is the Wassermann test of the nervous breakdown. A few well chosen questions should, however, result in establishing the differentiation between neurasthenic and toxic goiter syndromes.

HEAT DISTURBANCES

It is not possible to have an increase in basal metabolism without having an increase in heat production. The patients whose metabolic rates reach the higher levels are naturally the ones who complain most of a sensation of warmth. They wear fewer clothes, sleep under lighter covers and avoid warm rooms and close places. They require more fresh air than the normal person, as their need of oxygen is greater. Hot weather is very depressing to them.

Most of the symptoms of toxic goiter can be explained on the basis of altered physiology. Considerable is known of the pathological physiology of exophthalmic goiter. When considering the diagnosis of exophthalmic goiter, if the given symptoms do not coincide with the known physiological changes, it is safe to assume that one is dealing with another condition. It is in connection with this increased heat production in toxic goiter cases that I have observed one of the distinguishing differential diagnostic features between the toxic goiter patient and the neurasthenic. The former is warm and has warm hands, often flushed, with well distended veins. The neurasthenic is often cold or has chronically cold hands, the pale, veinless type. I know of no single differential diagnostic feature that is more significant than this. I have made it a rule to shake hands with every new patient that comes into the office. It has given me much valuable information relative to the line of questioning.

SWEATING

The human body, like the engine in an automobile, rids itself of excess heat by surface radiation and evaporation of mois-

ture. The sweating that these people complain of is nature's means of maintaining the body temperature at 98.6°F. in the face of increased heat production. Sweating is abnormally increased by both exercise and excitement.

MUSCULAR SYMPTOMS

Mention has already been made of tremor which is a neuromuscular symptom. Weakness is present in varying degrees, usually dependent upon the severity of the disease. In some cases it is the outstanding feature. As pointed out by Plummer, weakness of the quadriceps muscle is a characteristic symptom of exophthalmic goiter. It is brought to the patient's attention more noticeably in climbing or going down stairs, or in stepping up into a street car. Often the patient has to pull herself up. There is also an abnormal softness of the muscles. Pathologically, there is fatty degeneration of the muscle fibers. This accounts for the frequent early dilation of the heart in these patients, whose muscles have become soft and flabby.

DISTURBANCES OF SLEEP

Many complain of insomnia. They are restless in bed and complain of bad dreams and nightmares, a fertile field for the psychoanalyst.

SKIN CHANGES

Flushing of the skin is frequent in exophthalmic goiter, less so in adenoma. It is a vasomotor response and has a double protective function. It is due to surface capillary dilation and serves to lessen the intraarterial tension of an accelerated circulation, also to bring a larger volume of blood to the surface for heat radiation. Itching of the skin and a diffuse rash are occasionally encountered in the more active types of exophthalmic goiter, probably toxic in origin.

Increased pigmentation occurs in both diseases, but is seldom complained of by the patient. It is likely to be diffuse in exophthalmic goiter and localized around the neck in adenoma.

CHANGES IN THE VOICE

Changes in the voice develop from one of three causes. In those patients not operated upon they occur in my experience most frequently in exophthalmic goiter. The voice in these cases is weaker and slightly more coarse. The patients state that it is weak and that they have a tickling sensation in the throat. They are constantly clearing the throat on account of excess mucus. I have seen no paralysis of a vocal cord in an unoperated exophthalmic goiter. The voice changes have occurred in patients with very hard glands also very active glands, as the two are frequently associated. The voice changes in these cases are largely due to congestive disturbances of the mucosa of the trachea and larynx. They disappear when the goiter is removed.

Next in frequency of voice changes are those due to paralysis of one recurrent nerve. Just about 50 per cent of the patients who have consulted me for recurrence following operations have given a history of temporary loss of the voice for periods ranging from two to eight months thereafter. Almost invariably these cases have had a partial or complete paralysis of one vocal cord. Often one can get a clue to vocal cord paralysis by asking the patient to say "e-eee" in a high pitched voice. Frequently they cannot do it.

A majority of recurrences have been unilateral and have occurred on the same side as the functioning vocal cord, a most important point when considering further operative treatment. Vocal cord paralyses have occurred in untreated cases but in only 1 has a complete cord paralysis existed. Voice changes due to tracheal pressure and narrowing of the trachea have occurred in a considerable number of the larger adenomas and in a fairly high percentage of substernal and intrathoracic goiters.

CHOKING OR PRESSURE SENSATIONS

Choking and pressure sensations have been complained of in about 10 to 20 per cent of the cases. They, too, have occurred in a much greater relative percentage of substernal goiters. In cervical goiters, choking and pressure have had little or no constant relation to the size of the goiter. These symptoms have been present in simple colloid goiter about as frequently as in toxic goiter.

DYSPHAGIA

Difficult swallowing has been complained of in all types of goiter, but not very frequently is it the chief complaint. When it has occurred as such, the causative lesion has as a rule been in the esophagus and of the nature of a stricture, cardiospasm or malignancy. Fluoroscopic examination with barium meal should be made in all cases with such a history.

DYSPNEA

Two classes of difficult breathing have occurred. One, a shortness of breath on exertion, is of cardiac origin. It may mean cardiac damage or an overworked heart. The other, a pressure dyspnea, is encountered in adenomas and malignancies and is due to narrowing of the lumen of the trachea. The sounds made by these patients on coughing and deep inspiration are pathognomonic.

MUCUS IN THE THROAT

In the literature I have seen no mention of an increase of mucous as a symptom of toxic goiter, but a large percentage of patients have it. The usual history is that it began simultaneously with the onset of other symptoms.

HEADACHE

Headache has occurred in about 50 per cent of the cases. It has been characteristic in type, that is, it is usually occipital and associated with an aching pain in the back of the neck. Historically the goiter headaches are rather distinct, in that patients give a definite or approximate date of onset. Others who had previously suffered from headaches name a date on which they became worse, and since which they have been more frequent and more severe. The relief of headache following removal of the goiter has been striking, it being the only pain complained of.

EYE SYMPTOMS

One might think that the displacements of the eyeball, the disturbances of the ocular muscles and the abnormal relationship of the eye to the lids would constitute one of the very disturbing phases of the disease, but such has not been the case. A surprising degree of exophthalmos often exists without the patient being aware of it. The symptoms complained of are, as a rule, a burning and irritating feeling in the eyes and an early fatigue from reading. Quite a number have commented on a sensation as if something were pushing the eyeball outward. A few of the more advanced cases have developed squints and double vision, due to disproportionate weakness of the ocular muscles.

THE GOITER

As stated earlier, few symptoms are referable to the neck. A relatively small percentage of the patients have noted the simultaneous appearance of their symptoms with the appearance of a goiter. Those who have made such an observation have, as a rule, had exophthalmic goiters. A relatively large percentage of exophthalmic goiter patients have noted indefinite changes in the gland, among which are increases in size and hardness of the goiter and increased pressure. The changes in adenomas associated with changes in function on the whole have attracted less attention. They have existed for years as a rule, the patients having become accustomed to them. The local changes in circulation, vascularity, etc., do not occur so quickly nor so extensively as in exophthalmic goiter and are, therefore, less noticeable. If

an adenomatous goiter suddenly begins to grow in a person past thirty-five years of age, and continues, it usually means the beginning of hyperfunction.

GYNECOLOGICAL DISTURBANCE

So far as I have been able to determine, there has been no rhyme nor reason in the responses of the female reproductive functions to the effects of toxic goiter. The following gynecological symptoms have been repeatedly encountered. Amenorrhea: One woman, aged twenty-two, did not menstruate for fifteen months. This symptom developed shortly after an unusually severe exophthalmic goiter. Several had amenorrhea for periods varying from three to eight months. Others have had menorrhagia and metrorrhagia. In some all symptoms have been exaggerated at menstruation; in others they have been unaffected. The tendency for toxic goiter to develop at or about the menopause is well known.

CONCLUSION

No one symptom is infallible as a diagnostic criterion. One must get a mental picture of the altered physiological responses to the presence of toxic goiter, and with a knowledge of the possible resultant symptoms, one can in any given case of toxic goiter build up a picture that will fairly accurately fit the disease.

TRAUMATIC OPHTHALMOLOGY

CONTUSION OF THE EYELIDS AND CONCUSSION OF THE EYEBALL*

CONRAD BERENS, M.D., AND RAY R. LOSEY, M.D.

NEW YORK

SINCE surgeons and physicians who have had little training in ophthalmology are frequently confronted with patients complaining of various symptoms because of an injury to the eye from a blunt object, it may be of interest and real value to consider what may occur when the eyeball is struck, and what the treatment of the various conditions should be.

CONTUSION OF THE EYELIDS

Most medical men are familiar with the marked swelling of the eyelids which may result from the slightest injury. This swelling is due to the fact that, in order to facilitate the motion of the eyelid, the skin is very loosely attached to the underlying tissues. The familiar "black eye" is attributable to the extravasated blood from which the hemoglobin has been set free. In the early stages, until the swelling begins to subside, the condition is best treated by cold compresses of a saturated solution of boric acid. In severe cases an ice cap may be used or a small piece of ice wrapped in cloth may be applied. In some instances, even without fracture, the eyelid of the other eye may show discoloration. If the nasal wall of the orbit is fractured, an attempt to blow the nose may force the air into the surrounding tissues. This condition is easily diagnosed by the crepitation noted when the eyelid is palpated. In this type of injury, a pressure bandage should be applied and the patient should refrain from blowing the nose. Further treatment consists in keeping the nasal passages clean and applying cold compresses to the eyelids.

INJURIES OF THE EYEBALL

Rupture of the eyeball is often caused by a blunt object forcing the eyeball against the bony orbit. Usually the rupture takes place in the angle of the anterior chamber and frequently involves the canal of Schlemm which is concerned with the filtration of the fluids within the eyeball. This canal is in the thin part of the external coat, and is unprotected externally by muscles or other binding tissues. The conjunctiva is so elastic that the lens of the eye may be forced out under it without rupture and appear as a swelling beneath the conjunctiva. If this occurs and the patient is seen early the lens should be removed. Prolapse of part or all of the iris may take place, which would demand surgical intervention. It is usually necessary to excise the prolapsed iris, thereafter inserting a superficial mattress suture in the cornea and episcleral tissue. The conjunctival wound is closed separately. If the vitreous also tends to prolapse, it should be cut away and the lips of the wound brought together by one or two mattress sutures. It is remarkable how much vitreous may be lost with the retention of useful vision. Although many of these eyeballs go on to atrophy or secondary infection, useful vision is retained in some, and consequently, enucleation should not be too hastily performed. To prevent infection the patient should be given an injection of tetanus antitoxin or some non-specific protein to increase non-specific resistance to infection.

If the blow directed against the eyeball is not sufficiently severe to produce rupture

This is the first of a series of articles on Traumatic Ophthalmology. The next will appear in an early issue.]

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of the sclera, many other injuries, commonly not suspected by any except ophthalmologists, may result. *Hemorrhage* frequently follows contusion, subconjunctival hemorrhage being the most usual. This appears as a reddish black discoloration of the conjunctiva which tends to gravitate below the cornea. In the early stages this condition calls for cold compresses; in the later stages hot compresses are used to hasten absorption of the blood.

Hemorrhage may also occur into the anterior chamber, filling it with blood, and sometimes blood may find its way into the cornea, producing a typical picture caused by absorption of coloring matter from the blood. The presence of blood in the anterior chamber frequently leads to increased intraocular tension or secondary glaucoma. This condition sometimes requires a paracentesis of the anterior chamber in order to reduce the tension.

A hemorrhage into the vitreous may take place, or a subhyaloid hemorrhage, that is, between the retina and vitreous, may occur. This type of hemorrhage changes position as the patient moves his head. Hemorrhages into the retina usually appear flame-shaped when superficial or more rounded when deeper; they may be subretinal causing detachment of the retina, or subchorioidal with a resulting detachment of the chorioid. Subchorioidal hemorrhages appear as dark, rounded masses extending into the vitreous, and usually offer a good prognosis. Treatment of these hemorrhages usually consists of rest of both eyes, use of atropine in the affected eye, application of ice or iced compresses in the early stages, and hot compresses in the later stages. Following this, a weak solution of dionine may be used, beginning with ¼ of 1 per cent in the affected eye at night and gradually increasing until a 10 per cent solution is used.

CORNEA

The cornea is often slightly abraded by contusion but is rarely ruptured unless previous disease or injury has thinned it. To detect abrasion of the cornea a 3 per cent dilution of fluorescein is instilled into the eye; after irrigation the abraded area remains deeply stained. This condition is usually accompanied by great discomfort and sometimes by severe pain, which is best controlled by the use of a 2 per cent holocaine solution instilled into the eye. Bichloride of mercury ointment, 1:5000, is introduced between the eyelids, and a dressing is worn until the area no longer stains. Owing to the possibility of infection of the denuded area the use of a dressing is most important.

If a corneal rupture with prolapse of the iris has occurred, the iris should be excised, atropine instilled, and bichloride of mercury ointment, 1:5000, placed between the eyelids. No stitches are used unless there is a large wound the edges of which do not approximate properly, when a superficial suture may be inserted in the cornea with a fine curved needle and No. 1 or finer paraffined silk. The conjunctiva may be dissected up from the limbus and brought down over the wound where it is held by mattress sutures on each side of the cornea, which engage the conjunctival flap and conjunctiva below the cornea.

Milder injuries may give rise to a low-grade traumatic inflammation of the cornea, called traumatic keratitis, in which the stroma of the cornea may be filled with leucocytes. Rupture of Descemet's membrane, recognized as gray streaks under higher magnification, may also occur.

IRIS

The iris frequently shows one or two notches at the pupillary margin due to a partial or complete rupture of its sphincter. This is due to the backward displacement of the iris, and the rapid dilatation which occurs from the injury. If seen early the pupil may be widely dilated but after the inflammatory signs have subsided the use of a ½ per cent solution of pilocarpine and massage of the eyeball will tend to restore it to normal. However, there may

remain a slight notching of the pupillary margin. This is seen best under high magnification. Dilatation of the pupil may occur without injury to the sphincter, due to the rapid stretching of the muscle which produces temporary paralysis, or to injury of the third nerve fibers in the region of the ciliary body. The same treatment as for rupture of the sphincter is of benefit in some cases. The iris may be torn away from its attachment to the ciliary body. This is called iridodialysis, and may be only a small tear, or the iris may be completely separated from its attachment. Little can be done for this injury except to use atropine. If the iris is torn away sufficiently to obstruct the light entering the pupil or if it permits the passage of light through the normal and through the traumatic pupil, a monocular diplopia may be induced, sometimes making it necessary to perform an iridectomy of the prolapsed portion. Iris tissue does not usually tend to reunite, but fortunately this injury most often occurs at the periphery of the iris and causes little or no discomfort. The condition is frequently accompanied by hemorrhage into the anterior chamber.

CILIARY BODY

The iris and part of the ciliary body may become detached by a rupture through the pectinate ligament at the angle of the anterior chamber. This may cause a deepening of the anterior chamber and usually results in some weakening of accommodation. After the early use of cold compresses, atropine and rest, hot compresses and exercises in reading fine print at different distances may help to reestablish accommodation. If the lesion affects the ciliary body, which, according to many authorities, influences the secretion of the intraocular fluids, the secretion may be diminished with a resultant lowering of the intraocular tension. Weakness of accommodation may also result from injury to the ciliary body without apparent rupture of the tissues and for this injury accommodative exercises may be of value in the later stages. For the persistence of lowered tension in the eyeball, hot compresses are usually beneficial and in addition the use of increasing strengths of dionine solution. Subconjunctival injections of 5 to 10 minims of normal or hypertonic salt solution once or twice a week after anesthetization of the conjunctiva with 3 drops of 2 per cent holocaine and 2 minims of adrenaline, 1:1000, might be of benefit in raising the tension.

LENS

The most common injury is a dislocation of the lens. This is due to a partial rupture of its suspensory ligament which causes tilting of one edge of the lens resulting in a shallow anterior chamber in this area. Iridodonesis (trembling of the iris when the eye is moved) may result and is due to the fact that the pupillary margin of the iris lacks the support of the lens. If the angle of the anterior chamber is sufficiently blocked by the vitreous and by the iris which is pressed forward, intraocular tension may be increased. In severe injuries the iris may be caught behind one edge of the lens; this is known as retroflexion of the iris. Dislocation of the lens into the anterior chamber where it is held by the iris may also follow a severe injury. This usually requires removal of the lens, owing to the fact that circulation of the intraocular fluids cannot be maintained, resulting in secondary glaucoma. The lens may also be forced backward into the vitreous chamber where it may be held by the partially ruptured fibers of the zonule of Zinn or may float free in the vitreous and be seen as a white mass when the head is bent forward, when it usually appears in the pupillary area. In some instances the lens may be brought into the anterior chamber by having the patient hold his head down and forward. Eserine is instilled while the patient is in this position. Later the lens may be transfixed with a needle and removed. Dislocation of the lens into the posterior chamber is the oldest operation

for the treatment of cataract and is called "couching." In some cases no marked impairment or apparent disturbance of vision occurs. However, since glaucoma seems more likely to affect this eye than the normal eye, the lens should be removed if it can easily be done.

Cataract frequently results from these injuries because of a partial rupture of the capsule of the lens which permits the entrance of the aqueous with power to dissolve lens fibers. This may result in complete dissolution of the lens, or the lens may become partially opaque in some areas. If the opacity involves the pupillary area it is usually advisable to extract the lens in older patients, and needle it in patients under twenty-five or thirty years of age.

THE CHORIOID

The chorioid frequently becomes ruptured owing to stretching of the eyeball, as the chorioid is firmly attached anteriorly and there is a firm attachment posteriorly around the optic nerve. The rupture frequently appears as a crescentic lesion between the optic nerve and the macula. In the early stages rest and atropine provide the only measures of relief for this type of injury. But owing to the fact that a hemorrhage usually occurs from injury of the chorioidal vessels, rupture is, as a rule, not diagnosed until later.

When the hemorrhage occurs beneath the chorioid, the chorioid and retina may bulge forward and the lesion appear as a brownish red tumor covered by retinal blood vessels protruding into the vitreous. Chorioidal detachment by hemorrhage offers a better prognosis than retinal detachment, in that absorption of the hemorrhage usually occurs, leaving very little if any disturbance of eye function.

THE RETINA

The retina as well as the chorioid may be ruptured, but because the former is more elastic, it ruptures less frequently than the chorioid. Traumatic detachment of the retina is extremely common; it should be treated by rest and cold applications in the early stages, with the head elevated if the detachment is below, and with the head on a level if the detachment is above. Subconjunctival injections of normal or hypertonic salt solution are of value in this condition. If reattachment does not take place, trephine of the sclera and sucking out of the subretinal fluids may be attempted. Unless there has been a tear in the retina, good results are sometimes obtained. We do not hesitate to operate several times, even though the detachment reoccurs. One should also consider the treatment of complicating focal infections, particularly those affecting the nose and nasal sinuses, as we feel that these conditions may influence the outcome in a case of traumatic detachment.

A grayish white opacity of the retina, known as commotio retinae, is frequently noted as a result of injury. This gray opacity occurs in the region of the macula which appears dark red in comparison with the surrounding edematous retina. Although the vision may be markedly affected at first, it usually improves rapidly. Sometimes atrophy of the rods and cones results; the pigment epithelium may proliferate into the retina and appear as brownish yellow patches scattered around the injured area. In some cases, there is a shallow hole in the region of the macula, the macula appearing deep red with a gray, slightly elevated edge of the retina surrounding it. Since this is probably due to edema and degeneration of the nerve element surrounding the macula, it is usually observed some time after an injury.

OPTIC NERVE

A light blow on the eyeball may set up a low-grade traumatic inflammation of the nerve giving rise to congestion of the nerve head with possible blurring of the margins. In these cases field studies usually show a slight enlargement of the blind spot, and in some cases when the intraocular portion of

the optic nerve is not involved, there may be a retrobulbar traumatic neuritis and enlargement of the blind spot may be the only sign which can be noted. Two of our patients injured by squash balls showed this type of lesion with a slight diminution of visual acuity. If the injury is sufficiently severe, the nerve fibers may be torn away, resulting in evulsion of the optic nerve. The nerve fibers are less elastic than the sheath, which possibly accounts for this anomaly.

TRAUMATIC ENOPHTHALMOS

Traumatic enophthalmos occasionally follows contusion of the eyeball and orbit. Sometimes it occurs immediately after the injury but usually several weeks elapse before it becomes apparent. The condition most frequently encountered is a cicatricial contraction of the orbital tissue or an indirect fracture of the rim of the orbit with depression of the fragment of the bone outward so that the volume of the orbit is enlarged. Several cases have been reported in which no fracture of the orbit could be demonstrated, and Oast,[1] examining a patient one hour after injury, found the eyeball definitely sunken into the orbit about ¾ in. Examination under general anesthesia revealed complete divulsion of the oblique check ligaments of the recti. There were no well-defined adhesions except at the margin of the optic foramen. These were considered a possible cause of the enophthalmos, another possibility being that the nerve might have become incarcerated in the optic foramen. Bard[2] believes that enophthalmos associated with miosis, following diplopia and amblyopia after traumatism, is the result of injury to the orbital nerves and not to the trunk of the sympathetic.

Treatment naturally depends upon the exact anatomical cause of the enophthalmos. To lessen the enophthalmos Seefelder[3] tenotomized the four recti which did not produce diplopia and left a fairly movable eyeball. A roentgenogram of the region should be made in order to determine whether or not the orbit has been fractured.

SYMPATHETIC OPHTHALMIA

Sympathetic ophthalmia has been reported following concussions and injuries of the eyeball. Uveal pigment has been used in the diagnosis of this condition but the complement-fixation test for uveal pigment described by Woods[4] in 1917 and modified in 1921[5] has been discarded by him as impractical except for research work, and the intracutaneous test for pigment hypersensitivity substituted. The normal suspension, as originally suggested by Woods,[4] was made for us[6] and eighteen tests have been made with negative results, which should be expected if Woods' findings are correct, as no case presented signs of sympathetic inflammation. Woods and Knapp[7] were the first to treat sympathetic ophthalmia by desensitizing injections of uveal pigment in patients showing increased intradermal sensitivity. They treated 5 patients with desensitizing injections, and their results were encouraging, but as yet they do not wish to draw conclusions. We have had no experience with desensitizing injections.

If sympathetic inflammation is due to an infection, as some believe, nonspecific protein therapy may prove as beneficial as uveal-pigment injections, and Verhoeff[8] has apparently had good results with nonspecific treatment.

SECONDARY GLAUCOMA

Secondary glaucoma may immediately follow an injury or may be delayed for months or years. Any trauma which disturbs the anatomical relationship so as to cause a shallow anterior chamber, or to obstruct the angle with a resultant blocking of the excretory channels, tends to produce glaucoma. An intraocular hemorrhage, especially if subchorioidal, frequently causes the retina and chorioid to be pushed forward in front of the effusion of blood, closing the angle of the anterior chamber by pressure from behind and causing a rapid rise of the intraocular pressure as a result of the sudden increase of ocular contents.

Many other types of injury tend to bring about an increase in intraocular tension. Among these are rupture of the cornea with entanglement of the iris, producing an obstruction of the filtrating angle. Dislocation of the lens in such a manner as to cause interference with the outflow of aqueous current may induce secondary glaucoma.

Iridocyclitis, which frequently follows an injury, may precipitate an acute attack of glaucoma at any time. Acute glaucoma, coming on soon after an injury, is more easily recognized as the condition is usually accompanied by intense congestion, pain, loss of vision, and haziness of the cornea.

However, we must not lose sight of that type of glaucoma which comes on insidiously after many weeks or months, unaccompanied by pain or discomfort, but causing a steady loss of vision. The treatment must first be symptomatic; the tension must be lowered or vision will be lost. But above all else we must correct the cause, if possible.

CONCLUSIONS

From our experience in treating injuries of the eye it would appear that eyes are sometimes too hastily removed. Useful vision may often be preserved in eyes which at first appear to be totally destroyed.

Contusion injuries of the eyeball and eyelids in the early stages are usually benefited by cold, in the later stages by heat.

Because of the possibility of infection of the denuded area after abrasion of the cornea, a dressing is most important until the abrasion has healed.

Many injuries of the eye must be and can be well cared for by the general physician or surgeon who will find the ophthalmoscope and some knowledge of the normal tension of the eyeball important aids in diagnosis.

Secondary unrecognized glaucoma may rob the patient of useful vision which should be preserved. If glaucoma results, an ophthalmologist should be immediately consulted, as this condition is one of the most difficult of all ocular diseases to treat successfully.

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THE IMPORTANCE OF EARLY TREATMENT OF RECENT FRACTURES OF THE NOSE*

WILLIAM WESLEY CARTER, A.M., M.D., F.A.C.S.

NEW YORK

THE object of this communication is not to advocate the early application of such treatment as has, to a large extent, been given broken noses in the past, but rather to caution against the measures that have been commonly employed, for they have often been the direct cause of disfunction and have frequently been responsible for deformities that might have been avoided, or at least have been less severe, had no treatment whatever been given at the time of the injury.

For some reason injuries to the nose have not received the attention that their importance deserves. This may be due to the fact that the injury to the nose of itself, seldom threatens the life of the patient, consequently both he and the doctor are content as soon as the hemorrhage has stopped and a piece of adhesive plaster has been tightly stretched across the bridge of the nose. The latter is about the worst thing that could be done to a nose that has been macerated by a blow. Strange to say this is the treatment usually received, as elicited from the patients coming to me for the correction of old traumatic deformities, many of whom are also suffering from pronounced nasal obstruction.

Unfortunately the resiliency of the cartilage and the swelling of the soft tissues often mask at the time of the injury the real damage that has been done to the supporting framework of the nose; it is this that determines the complications that so frequently follow. It is the duty of the one who first sees the patient after the injury to take steps to avoid these disagreeable sequelae. He should bear in mind that the immediate results are of far less importance than those that will follow later if the case is not properly treated.

Inspection, on account of the swelling, may reveal nothing of value, and one may be deceived by palpation, as the crepitus of both blood and emphysema closely resemble that of fracture.

In children roentgen-ray examinations are usually unsatisfactory. In adults not only must a soft tube be used but the position of the head in making the plate must be just right in order that a correct estimate of the lines of fracture can be made and the displacement of the fragments can be correctly judged. Roentgen-ray examinations of injuries to the nose give one an idea of their extent but it is difficult to estimate the exact displacement of the fragments by this means because the position of the head varies and, furthermore, the delicate shadows cast by the parts involved leave on the plate only an indistinct and incomplete record of what has happened. In some cases it is remarkable, as shown by the roentgen-ray plate, the distance fragments of the nasal bones will be displaced from their original position by a severe blow upon the nose. Because the fragments are not acted upon by muscular traction, as is the case in fractures of the long bones, it is not easy to explain the phenomenon. This inability to follow up the lines of force which produced the displacement adds to the difficulty of replacing these fragments in their proper position for, the direction of the force being unknown, it is not possible to correct the deformity by reversing it.

The importance of apparently trivial injuries to a child's nose is frequently not realized until years later when a badly

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deflected septum and a distorted or flattened nasal bridge can be traced back to an injury which at the time received

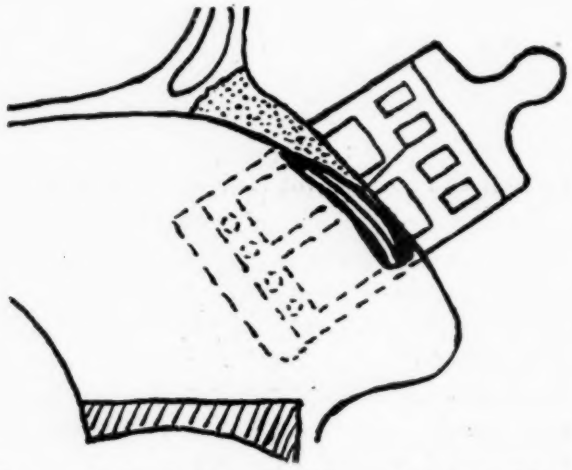

Fig. 1. Sectional view of bridge splint in position.

inadequate or no treatment at all. The frequency of this catastrophe, as noted among the histories of the patients who come to me for correction, prompts me to say that all traumatic injuries to the nose before the organ is fully developed should receive careful consideration at the time and be given treatment intended to forestall the complications that so frequently follow later in life.

The diagnosis of fracture, as I have already said, is not always easy, especially in children. Therefore after utilizing all of the aids to diagnosis already mentioned, including roentgenography, if we are still in doubt as to whether or not there is a fracture but are inclined to believe that there is on account of the distortion of the organ, the patient should be given the benefit of the doubt and proper treatment for fracture instituted.

This view of the situation may seem ultra-conservative to those who are not interested in this special field and who are not having brought to their attention almost daily cases that bear indisputable evidence of neglect or maltreatment of injuries to the nose in early life. This situation prevails with all of us as regards conditions outside our own field of effort. We should realize this weakness that ever threatens specialization and try to keep ourselves in touch with the other fellow's work, at least to a degree that will enable us to make a reasonable diagnosis and offer advice as to whom he should consult for the relief of his particular ailment. It is unnecessary for me to say that a man who has first practiced general medicine and surgery before taking up a specialty is far better equipped not only for successfully practicing in his own specialty, but for recognizing the relationship between his own work and that of specialists in other fields. The judgment of a man who goes into a specialty immediately after leaving college is always warped, even though he may become eminent in his own field. Many of his mistakes and clinical failures, however, may be attributed to his unfamiliarity with conditions that fall within the ken of the general practitioner or specialists in other fields to whom he might refer them with advantage to the patient and credit to himself.

The point the writer wishes to make is that traumatic injuries to the nose should receive early attention from one skilled in diagnosing the extent of the injury and one who is accustomed to treating such cases with a view to preventing future trouble.

The treatment of recent fractures of the nose depends chiefly upon the age of the patient, the extent of the injury and the time that has elapsed since the injury was received. Very seldom does it happen that the case is seen immediately after injury and before there is time for swelling to occur. If the swelling is great, no attempt should be made to set the fracture or to place the dislocated parts in their proper position; this cannot be done accurately at this time, for the extravasated blood and general swelling cause wide separation of the segments of the arch.

The nasal cavities should first be cleansed with Dobell's solution, and the outside cuts and abrasions painted with tincture of iodine, followed by alcohol. Alternate cold and hot compresses or my

nasal water jacket should then be applied until the swelling subsides. On account of the very great rapidity with which healing occurs in this locality, the parts should be adjusted as soon as possible. Healing, following an injury, occurs more quickly in the nose than in any other part of the body; we may expect fairly firm bony union in twenty days. Roentgen-ray examination, too, should be delayed until swelling has subsided. In the case of adults, roentgenography may be of some value in determining the extent of the injury and location of the segments, if fracture or dislocation has occurred.

The management of the case depends mainly upon the extent of the injury and the age of the patient. The use of the bridge-splint in the young is, for obvious reasons, impracticable. Here we must, by the sense of touch and by the use of a small size moulding forceps, such as my own, endeavor to replace the fragments in their proper position. One blade of the forceps is introduced into the nasal cavity and the other, padded with rubber tubing, rests on the outside of the nose. The septum is lined up by placing a blade on either side and simultaneously the fragments are manipulated and a slight lifting pressure towards the dorsum of the nose is exerted.

After adjusting the parts, the writer ntroduces into each nasal cavity a gold wire splint which he has perfected. This is made at the time to suit the case. These are usually left in for forty-eight hours.

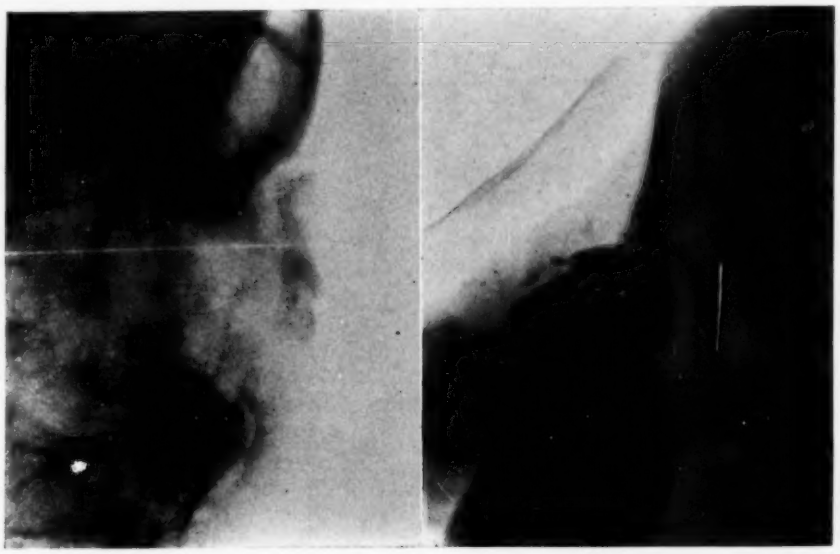

Fig. 2. Fig. 3.

Fig. 2. Fractured nose with upward displacement of both nasal bones.
Fig. 3. Compound comminuted fracture of nasal bones.

Fig. 4. Fracture of bone transplant two years after operation. (This case was treated with the bridge splint. The broken ends of the transplant became firmly reunited, with entirely satisfactory result.)

In some of these cases in which there is a tendency of the fragments to protrude or override, it is necessary to apply tin-

Fig. 5. Fracture of tip of nasal bones.

lined copper splints also perfected by the writer, to the outer side of the nose.

In treating children we must keep constantly in mind the fact that the nose has not yet reached adult proportions and that its normal development depends upon the accurate adjustment of the fractured and dislocated segments of the nasal arch and the lining up and retention of the septum in a vertical position. This is of paramount importance, *for the normal elevation of the nasal bridge depends upon the growth of the septum.*

In fractures of the nose in adults the developmental factor may be ignored. Here we consider the nasal arch more from a mechanical point of view. For this reason we may find most useful the bridge splint which the writer devised in 1908 and which, in selected cases, is used with great satisfaction. It is intended to duplicate the forces employed by nature in the development of the nose. It consists essentially of a hinged, adjustable bridge which fits over the nose and two intranasal splints attached to sutures which pass through the dorsum of the nose and are tied together over the hinge of the bridge after the apex of the nasal arch has been lifted to its normal elevation. The writer has elsewhere described in detail the appli-

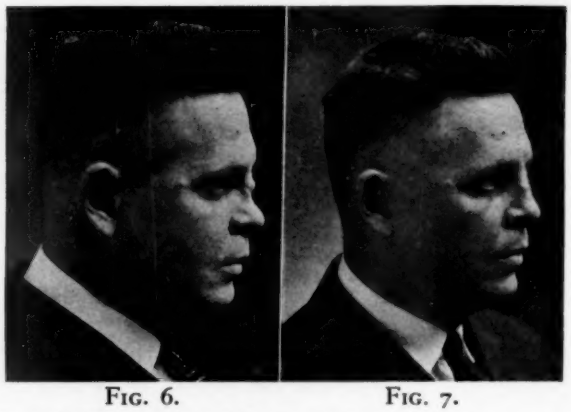

Fig. 6. Fig. 7.

Fig. 6. Old compound fracture of nasal bones.
Fig. 7. Case of Figure 6 after correction by subcutaneous transposition of tissue by intranasal route and bone transplantation.

cation and use of this instrument, which, when properly applied before faulty bony union has occurred, will pull the macerated, displaced segments of the nasal arch back into their proper position and hold them there until union has occurred. It is well not to have the traction on the sutures attached to the intranasal splints any greater than is necessary to raise the depressed fragments into proper position. One must also take the precaution to pad well the edges of the wings and to loosen up these each day by means of the adjustment screw and bathe the skin upon which these rest with 95 per cent alcohol. The bridge of the nose where the sutures emerge should also be bathed with alcohol frequently and the nasal cavities should be cleansed with Dobell's solution.

Fractures of the nasal bones probably always communicate with the nasal cavities through lacerations of the mucous membrane, and frequently the fracture is compound through the skin. In the latter the fragments may be more accurately adjusted through the open wound. The writer has, on several occasions, been obliged to enlarge the wound in order to accomplish this, and at times has found it necessary to suture the fragments in place

with chromic catgut. These open wound cases are not suitable for the bridge splint. In these compound cases not only should every antiseptic precaution be observed and the fractured segments brought into proper position, but in suturing the skin wound an effort should be made to bring together the edges of the fascia which lies beneath the skin and which prevents the latter from becoming adherent to the underlying bones. If this is carefully done, the scarring will be much less noticeable as the indentations due to union of skin to underlying bone will be avoided. The ideal repair in these cases leaves the skin movable over the underlying nasal bones.

The following conclusions are drawn from clinical experience:

1. All injuries to a child's nose should receive careful attention and should be treated with a view to conserving its developmental forces, especially those inherent in the septum.

2. Nasal fractures in adults, especially if depressed, are best treated by means of the bridge splint.

3. In compound fractures the fragments should be brought into proper position by means of the moulding forceps and the skin wound so sutured that the fascia remains movable over the nasal bones.

SURGICAL CONSIDERATION OF THE CERVIX UTERI*

RAFE C. CHAFFIN, M.D., F.A.C.S.

LOS ANGELES

ANATOMY AND HISTOLOGY

THE cervix is that portion of the uterus from the external os up to its junction with the corpus uteri, 1 to 1½ in. long. It is made up of muscle tissue which is continuous with that of the body of the uterus and is subject to and capable of undergoing all physiological and pathological changes that are found in the uterine body. The canal is normally lined with columnar epithelium and below this layer in the substance of the muscle tissue are numerous mucus-secreting glands. Normally, the secretion is sufficient to keep the canal and vaginal mucous membrane moistened and to prevent friction of the vaginal walls in their collapsed state. The cervix is abundantly supplied with sensory nerves derived principally from the sacral and lumbar plexes. The blood supply is derived from the circular artery which itself is a branch of the uterine artery at the termination of the internal iliac. The lymphatics of the cervix drain into the lymph vessels of the broad ligament and communicate freely with those of the tubes and body of the uterus and lie between the folds of the broad ligament. Like all smooth muscles the cervix is elastic and contractible. The important physiological changes taking place are those which occur during pregnancy and following delivery.

PATHOLOGY

INFECTION. Infection furnishes by far the greater number of pathological cervices coming under our notice. The invading organism may be of any of the pyogenic group and, less frequently, is that of diphtheria, tuberculosis or syphilis.

The mechanism of the infection is an interesting one. The organism gains entrance to the vagina through coitus, douches, instruments and by continuity (colon vaginitis) and possibly by bathing. There is no age limit, infection being seen in the young nursing infant and in the aged. The invading organism, gaining entrance to the cervical canal, causes a swelling of the mucous membrane and if not destroyed rapidly finds its way to the ducts of the glands and, because of the swelling of the epithelium of the ducts, the glands no longer function but become potential abscesses.

In the milder cases the ducts are not completely occluded but continue to drain incompletely, resulting in infiltration, an increase in fibrotic tissue and hyperplasia, and hypertrophy of the mucous membrane of the canal and glands. This mild form is seen in infants, young girls and the virgin. This type of case concerns us mostly from the standpoint of mild leucorrhea, dysmenorrhea, menorrhagia, metorrhagia and reflex pain along the origin of the cervical nerves or as a focus of infection with few or no local symptoms.

The more serious infections are those of gonococci, staphylococci and streptococci usually resulting from instrumentation or trauma. The mechanism of invasion here is similar but all pathological changes are more marked and the extent of the infection depends either on its early control or the resistance of the human organism. In more advanced cervicitis, the cervix is bluish and enormously hypertrophied, oftentimes to 6 cm. in diameter, exudes quantities of mucus and is smooth to visual examination but decidedly nodular to the examining finger (Fig. 1).

On the surface are seen numerous thin,

*Submitted for publication June 25, 1928.

glistening areas which are the large distended Nabothian cysts, formerly the deep cervical glands. Some may rupture from time to time only to refill in a few weeks. Each and every occluded gland may contain live organisms, as can be easily demonstrated by smears and cultures. Pus or infection under pressure results in one of two things: either the patient's resistance is so high that the bacteria are eventually destroyed or rendered non-toxic, or the patient suffers from toxemia or extension of the infection by way of the lymph channels to more remote areas, causing metritis, salpingitis, perimetritis, peritonitis, etc.

There is also a group of cases in which is found the picture just described but without definite symptoms of infection, at least as far as can be determined by ordinary examination, for the mucus is sterile. These may, however, and often do, present the symptom of pelvic pain or low backache at the origin of the nerve supply of the cervix. I have often proved this point by simple puncture, which relieves the symptoms temporarily. The pain seems to be due to local pressure of the distended gland on the nerve endings to the cervix. Our further proof comes from the patient as well as the physician who states that curetting and repair of the laceration relieves the patient of pelvic and low back pain. I attribute this relief entirely to a temporary improvement in the infection by curetting the cervix and compressing the glands by the mechanical dilator or, in the repaired cervix, to the accidental or intentional puncture of the cysts or the prevention of absorption through the everted and exposed cervical mucous membrane. Women will often have relief after puncturing the cysts and will return for a repetition of the treatment, knowing when they have become occluded. I might mention here that investigators do not agree as to the route by which cervical infection reaches the pelvic organs, whether by the uterine canal or lymphatic channels, but at the moment it seems not to matter, for all admit that the cervix must be first infected, and that is the area with which this paper is concerned.

Trauma. The cervix is traumatized

Fig. 1. Lacerated cervix with almost complete degeneration of cervical tissue. Retention cysts seen both deep and superficial.

by childbearing, mechanical dilating, instruments, curetting, pessaries of various sorts and foreign bodies in attempted abortions. The most frequent and by far the larger number are the lacerations at childbirth (Fig. 1). This leaves ragged abrasions of varying depth and number in the cervical tissue, exposing both the cervical mucous membrane and the injured glands themselves. This exposes a fertile field for the entrance and propagation of pathological organisms and although the symptoms are not particularly evident a closer examination and investigation will elicit such resulting abnormality as (1) subinvolution, (2) chronic discharge or (3) prolonged lochia flow, low back and sacral pain, menorrhagia and broad ligament tenderness and pain.

If the immediate torn surface is untreated, it is covered by scar and vaginal epithelial tissue but the lips become everted and hypertrophied and expose the delicate endocervical mucous membrane to the many infectious organisms found at all times in the vaginal vault.

New Growths. The types most frequently seen under this classification are cancer and fibroma. We need not go into detail regarding cancer of the cervix, except in regard to its prevention. I think it may be said without argument that very few cervices that have not been subjected to infection or trauma become the seat of primary cancer. This point of prevention will be emphasized under treatment.

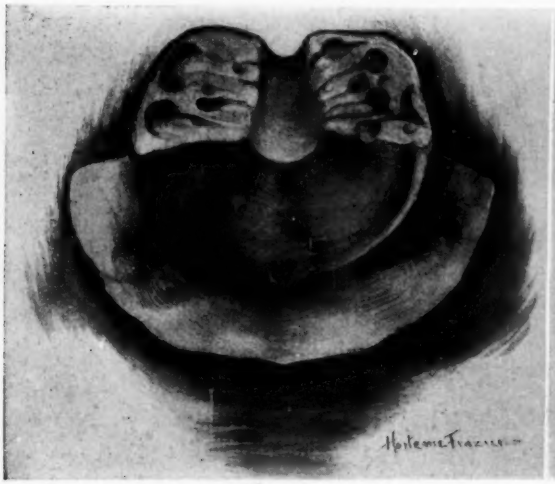

Fig. 2.

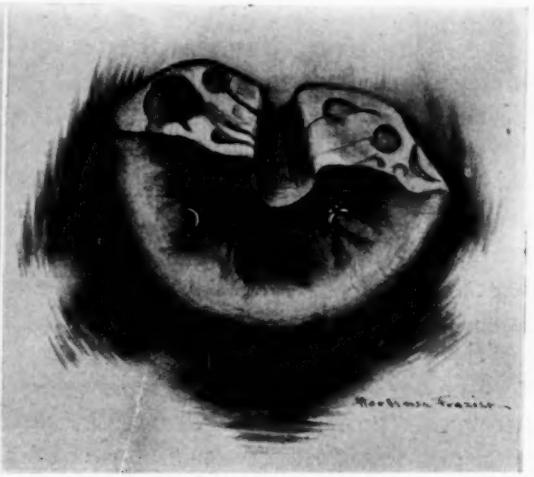

Fig. 3.

Figs. 2 and 3. Show that many of the coring amputations are not sufficiently high to reach the deeper glands.

The cervix is fibrous or fibroid depending upon whether the fibrous tissue is diffuse or circumscribed. The fibrosed type has been completely described under chronic infection. The fibroid cervix may vary from a slight ½ to 1 cm. in diameter, nodular in the canal and usually on the anterior lip, to the large fibroma completely filling the true pelvis. These are each worthy of serious consideration. The small nodule in the musculature of the cervical wall or beneath the mucosa as a polyp usually is manifested symptomatically by menorrhagia and abnormal mucous secretion. It is easily reached and the technique for removal is quite simple. The large growth is difficult to deal with because of the inaccessibility and because of the possible distortion of the anatomy of the pelvis. It is in this type of fibroid that we so often find the ureters dislodged from the posterior position and stretched across the superior surface of the tumor. If this condition is not recognized injury may result and even then the tumor may be so large that its removal from beneath may become extremely difficult.

TREATMENT

The catarrhal cervix as found in the very young is the only condition which may be treated medically with success. But it is in this particular type of cervix that medical treatment is most difficult because of the inaccessibility of the infected tissue. When it can be exposed, chemical cauterant with astringent and penetrating antiseptics are frequently curative. Diathermy has recently been applied with some degree of success. The actual cautery, grossly applied, is of value, although it should be used with great caution for fear of extensive destruction of the mucous membrane, which might possibly cause sterility and not infrequently does cause stenosis or occlusion. The knife cautery is of value and causes much less destruction of tissue but for the same reason is not as efficient as the Percy cautery. The knife, Post and other small cauteries fail in their effectiveness principally because they cool too quickly and do not carry sufficient heat to the high and deep tissues to destroy the deep glands (Fig.

11). Fulguration has been most satisfactory in my hands, and I shall explain the technique in detail later.

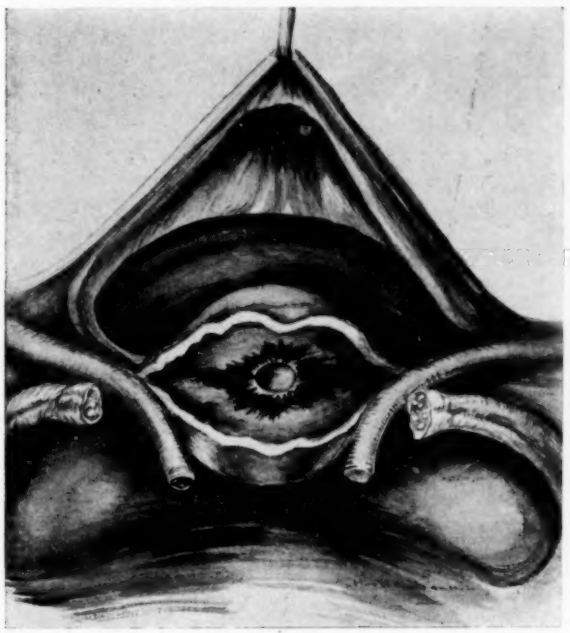

Fig. 4. Cervix cauterized from above with Percy cautery immediately after amputation of corpus.

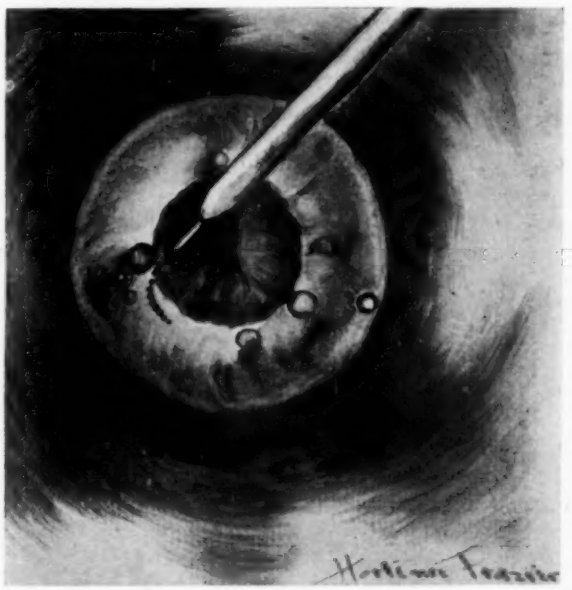

Fig. 5. Shows manner in which fulgurating electrode can be introduced into deep glands without destruction of the surface.

A complete cure is effected when the cervix has returned to normal size and consistency and when the secretion is clear and transparent and not of sufficient quantity to cause an uncomfortable amount of moisture at the vulvar orifice. Many times the number and size of retention cysts are out of all proportion to the amount of excessive secretion noticed by the patient. All cervices should be carefully examined regardless of whether the patient states she has an excessive secretion. The more advanced type of cystic cervix seen in the non-virgin requires more drastic treatment.

In the beginning of the study of the patient, if we are seeking a focus of infection,[1] it is permissible to make numerous stab or puncture wounds deep into the substance of the cervix and express cyst contents with the aid of the ordinary sponge forceps. One ring blade is placed in the canal and the other at several successive points on the outside of the cervical canal and pressure applied, which maneuver will express quantities of mucus or mucopurulent material. If this gives definite relief to the patient for a certain pelvic discomfort and low back pain, the examination should be carried still further by smears and cultures made from the gland contents. The puncture treatment, however, is not curative and more radical measures must be applied to prevent the reformation of cysts.

After carefully considering all forms and methods of treatment, it seems that the curative agent must be either some form of heat or resection. I have classified the fulguration current with the cautery. Amputation, the most radical form of treatment, also probably is the surest means of eradicating the infection but this necessitates hospitalization and a considerable period of disability. Any of the several plastic operations may be done, depending somewhat on whether or not the cervix is lacerated. In the lacerated, infected and cystic cervix, probably either the Emmett or Schroeder amputation is the method of choice. At this time I want to emphasize the importance of carefully examining the apparently simple, lacerated cervix for

deep retention cysts before doing the classical denudation for repair. This may be done by numerous stab wounds into

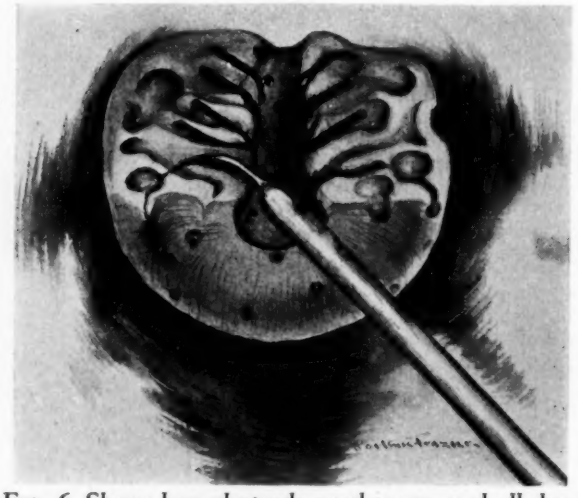

Fig. 6. Shows how electrode reaches any and all deep glands regardless of depth or size.

the cervical tissue and if one deep-seated cyst is found there are probably more and the repair, if done, will probably not be satisfactory either cosmetically or symptomatically. The Sturmdorff coring operation has been, in my experience, the most satisfactory of all surgical treatment in the non-lacerated cystic cervix. It does not, however, produce a complete cure, mainly because the dissection often cannot be carried sufficiently high to remove all glands (Figs. 2 and 3). Incidentally, I have first-hand knowledge of five cases of stenosis of the canal following this operation, two of which were my own. It was found necessary to dilate them from one to several times over a period of months, but all were eventually cured as soon as the canal epithelization was complete.

In our service at the General Hospital and in private practice, we have tried out the Percy cautery on a series of several hundreds of cases. These patients are all in the hospital for surgery, usually for hysterosalpingectomy or salpingectomy alone. Some few are in for repair of the outlet and, if no evidence of adnexal disease exists, the cystic cervix is treated with the cautery. The technique is that of placing the patient in the lithotomy position with no field preparation, with weighted speculum, two Sims' lateral

Fig. 7. Inserting the electrode into the cervical tissue.

retractors, a tenaculum and cautery with the round straight point heated to redness without the rheostat. The point is thrust well up in the canal from three to five times for one to two seconds each. It is better to withdraw and reinsert so the point of the cautery farthest away from the heating element can again become hot; it cools rapidly in the tissue. Usually the cauterization should not be so radical in those patients retaining the corpus uteri as in hysterectomy patients.

For the past five years I have adopted a technique in subtotal hysterectomies of using the cautery always from above (Fig. 4). This is my rule, even though I have done a pre-operative cauterization from below. The technique is that of plunging the tip of the red-hot instrument down into the canal immediately on

amputation of the corpus. It is simple, clean and takes no more than a few seconds longer. (If not ready, a cautery will heat to a sufficient degree in three minutes.) This method controls bleeding, sterilizes the field, destroys all mucous membrane and deep infection, and in no way complicates the technique. Since using it I have had no "come back" of discharging cervices; I had many before its use and have cauterized several of these since. In my clinic at the General Hospital and in my private practice in the past year, I have seen 15 or 20 cases in which subtotal hysterectomy had been done, and the patient came in for relief of a profuse vaginal discharge. Had the cautery been used at the time of operation, the patient would have been saved the annoyance and inconvenience of retreatment and some danger of cancer.[2,3]

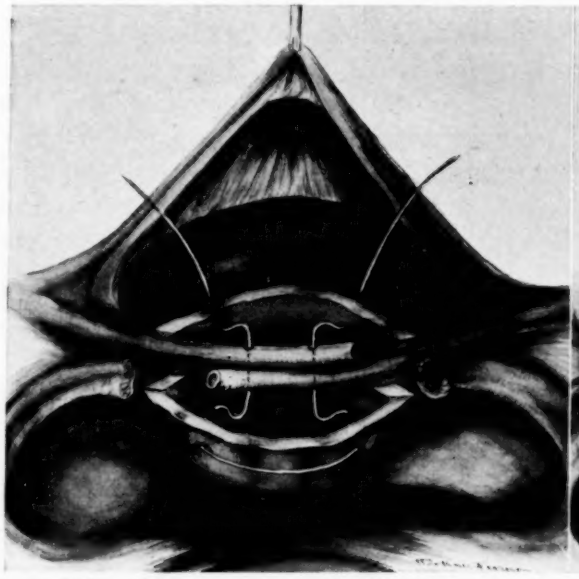

Fig. 8. Subtotal hysterectomy. Chromic catgut suture placed after amputating and cauterization of canal.

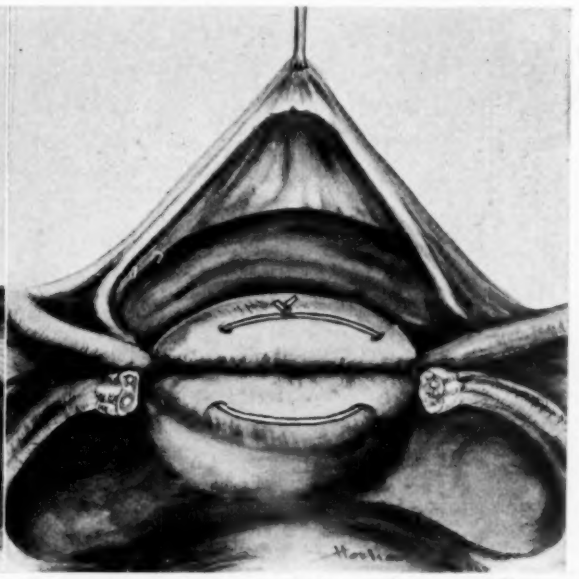

Fig. 9. Mattress suture tied, crossing round ligaments and closing lips of cervix.

Fulguration. My experience with this treatment covers about two and one-half years and between 200 and 300 cases. All cases have been followed up as far as it is possible to get the patient's cooperation. They are examined at two, occasionally four, six and eight weeks after treatment. The patient is usually not benefited under four weeks, but at the sixth or eighth week's visit they report that they are well as far as they know, and a similar report is obtained in at least 95 per cent of the cases treated. With few exceptions, treatments have all been done in my office and in the Out-Patient Gynecological Clinic at the General Hospital. The majority are treated without anesthesia, a few with gas-oxygen analgesia and some with novocaine.

Technique. Insert a wide short bivalve speculum and draw down the cervix with a hook in the canal (the hook is less painful than the tenaculum). If local anesthesia is used, infiltrate with 1 per cent novocaine around the periphery of the cervix under the vaginal cervical mucous membrane; no intracervical or canal infiltration seems to be necessary. The machine is set for 300 to 500 milliamperes which seem to be the average amount necessary, and the current is applied for from two to five seconds, except that in treating the cavity of a large cyst a longer application is necessary. All surface cysts are attacked by direct puncture, as in the use of the knife (Fig. 5). The canal and deep invisible areas are treated by the electrode from the

canal outwards, usually to a depth of ½ cm. (Figs. 6 and 7). As many as twenty or thirty stabs may be made in a few minutes

Fig. 10. Illustrates graphically the method of preparing cervix for suspension after cautery of canal.

time, and cause the average patient very little discomfort.

The patient is able to leave the office or clinic immediately, and many working women have returned to duty at once without loss of time. They are all told that the discharge will increase immediately and be blood-stained, and that no improvement is to be expected for from two to four weeks. I insist on the patient remaining in bed for two to three days during the following menstrual period because of excessive menstruation in a few patients. This is, however, no worse than is expected after any surgery on the cervix. The patient is asked to return after an interval of two weeks for examination in order to note the extent of the slough, the shrinkage of the cervix, menstrual bleeding, etc. A few cases have required two or three treatments but these had large cysts, some containing nearly a dram of fluid. It is probably much better to repeat the treatment than to attempt too much cauterization at one time, as by the latter method the slough is greater and the danger of hemorrhage increased.

The results are obtained by tissue

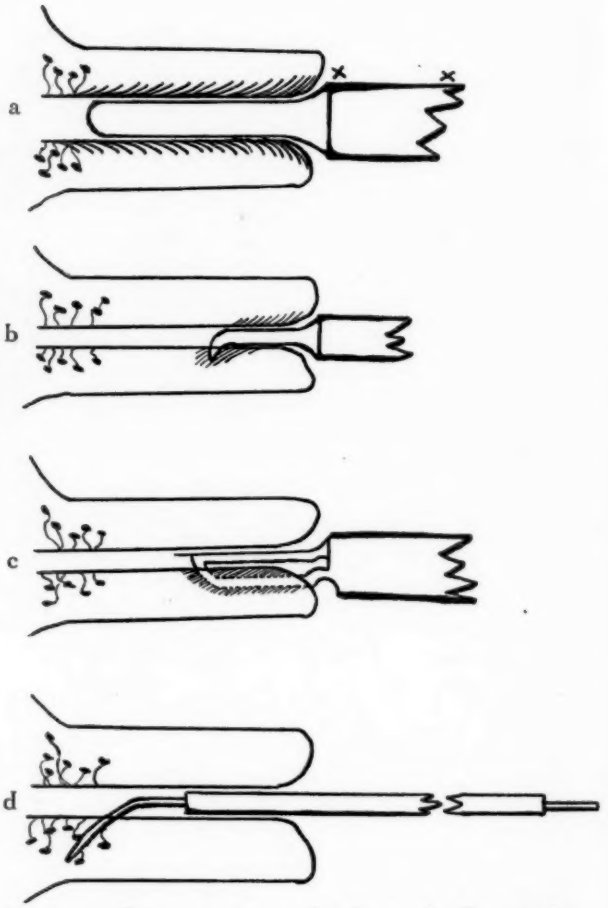

Fig. 11a. Percy cautery, the best of all cauteries. Heating element at x-x. Not sufficient heat at tip; massive destruction at external os. Shaded area indicates location and penetration of heat.

b. Post cautery. Insufficient heat, does not reach higher glands. Moisture cools point rapidly.

c. Knife cautery. Does not reach high glands. Cools quickly.

d. Fulguration electrode, no limit to height or depth of application. Degree of destruction under perfect control. The only cautery which reaches the deep glands.

Four diagrammatic drawings illustrating effects of different kinds of cautery. The shaded area in the drawings illustrates the location and penetration of heat. The only one of the four reaching the deep glands is the fulguration electrode.

reaction rather than actual destruction of the cyst areas. It seems that the advantages of fulguration over the cautery are due to the surgeon's being able to localize the agent and to apply it to considerable

depth without destroying all intervening tissue. Also the mucous membrane of the cervical canal is not completely destroyed, permitting of complete reepithelization of the canal, thereby avoiding stenosis. Five or 6 of the cases treated have had small (½ to 1 cm.) fibromas in the anterior lip of the cervix, and these were easily destroyed by inserting the electrode down to the center and applying a somewhat stronger current, much as a wart or mole is treated with this agent.

There is one more point relative to surgery of the cervix that should be emphasized, and that is adequate suspension following the subtotal hysterectomy and cauterization. It is not rare to find patients with prolapsed cervix following hysterectomy. I can highly recommend a technique I have used for the past six years which I term the "cross over mattress technique" (Figs. 8, 9 and 10). I have not been able to devise any more adequate and at the same time simpler and more easily performed method for my hysterectomies. One double No. 2 chromic gut mattress suture secures and buries the crossed ligaments between the anterior and posterior lips of the cervical stump. A simple plain suture peritonizes. I have had the opportunity of seeing a number of these cases afterwards and the pictures were all like the one illustrated here (Fig. 9). The ligaments had grown into the uterine musculature as firmly as they were ever implanted in the cornua by nature.

CONCLUSION

1. The cervix may be the seat of concealed infection for which the tonsils, teeth and many other fields have been blamed but the fact not demonstrated.[4,5]

2. The cervix should always be carefully examined and the condition noted even in the absence of pelvic symptoms.

3. All pathological cervices should be treated adequately and early.[6]

4. In the removal of gross pathology we should not forget that the cervix probably took some part in the etiology and should be treated with major surgery. If this point is kept in mind, the patient will return for final examination completely cured.

5. The Percy cautery is probably by far the best treatment for the cervix in subtotal hysterectomy and should always be used.[7] By using it as recommended, a complete hysterectomy is seldom necessary except possibly in malignancy. The mortality rate in total hysterectomy is much greater than that in subtotal hysterectomy.

6. Leaving the cervical stump is of definite value to the patient in preventing vaginal distortion and in retaining the normal pelvic diaphragm, and if properly suspended the incident of the vaginal hernia should be little if any greater than when the corpus uteri is present.

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ACUTE TRAUMATIC CRANIOCEREBRAL INJURIES

GRAPHIC CHART USED FOR TEACHING*

I. M. GAGE, M.D.

NEW ORLEANS

IN the treatment of head injuries it is necessary thoroughly to understand the underlying pathology responsible for the clinical signs and symptoms in order that we may anticipate processes in advance of their appearance, that sound therapeutic measures may be instituted to restore the individual to normal or prevent disaster.

Years ago a patient with a fracture of the skull was treated for fracture of the skull only. Today, however, we know that the underlying cerebral injury is responsible for the different clinical pictures. Most, if not all, of the symptoms following head injury are due to increase in intracranial pressure. Whether it be due to hemorrhage (extra- or subdural), increased secretion of cerebrospinal fluid, decreased absorption of cerebrospinal fluid, or a combination of two or more of these conditions, the results are the same: increased intracranial pressure.

The research work of such men as Cushing and his coworkers, Frazier, Sachs, Sharp, Coleman and Dowman, has stimulated widespread study, with subsequent thorough understanding of head injuries and the institution of sound therapeutic measures for their relief.

The treatment of acute craniocerebral injuries today is that of conservatism. First the patient is treated for shock, if present, by the usual methods applicable to the treatment of this condition wherever encountered. When the patient has recovered from his shock, dehydration is indicated to combat the development of increased intracranial pressure. Dehydration is produced by the administration of hypertonic solutions intravenously and a hydragogue cathartic given by mouth or rectum. This is supplemented by operation when indicated.

In teaching students the pathology of cerebral injuries and the underlying changes responsible for the clinical phenomena found, it is difficult to portray by word pictures the different pathological changes, the pathological physiology, the most appropriate treatment, and the time for its institution. To surmount these difficulties, the accompanying chart was constructed. It has been gratifying to find that students who use this chart have a fair knowledge of the pathology of head injuries and the fundamental indications for treatment.

EXPLANATION OF CHART

For convenience we have divided the clinical picture of head injuries into six stages, representing the phenomena produced by the increase of intracranial pressure with subsequent changes in pulse rate, blood pressure, and cerebrospinal fluid pressure.

STAGE 1. This represents the stage of shock, when every effort should be made to secure the return of the patient to normal by therapeutic measures, i.e., modern treatment for shock. If intravenous infusion is indicated, we prefer to give a 50 per cent solution of glucose, which produces an increase in the blood volume by virtue of the bulk of the solution itself, as well as by dehydration from the tissues into the vascular tree. It will be noticed that in Stage 1 there is a fall in blood pressure and an increase in pulse rate, the spinal fluid

* From the Department of Surgery, School of Medicine, Tulane University, New Orleans.

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pressure, however, being above normal. In severe craniocerebral injuries the patient rapidly passes from Stage 1 into Stage 6 in determining the spinal fluid pressure and the character of the spinal fluid, whether clear, bloody or xanthochromatic, and

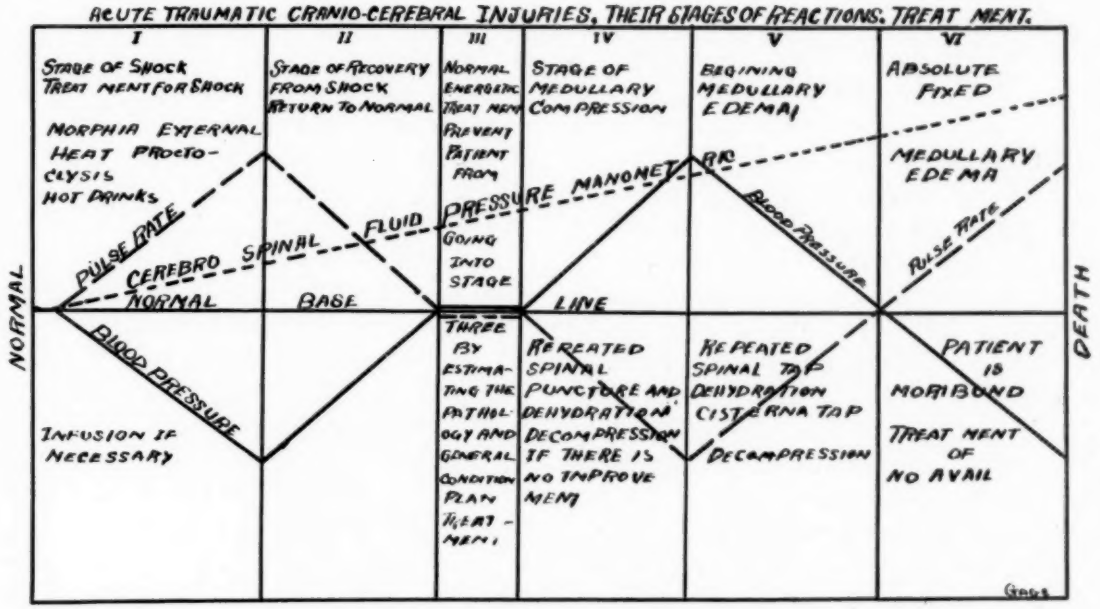

ACUTE TRAUMATIC CRANIO-CEREBRAL INJURIES, THEIR STAGES OF REACTIONS. TREAT MENT.
I
STAGE OF SHOCK
TREAT MENT FOR SHOCK
MORPHIA EXTERNAL HEAT PROCTO-CLYSIS HOT DRINKS
II
STAGE OF RECOVERY FROM SHOCK RETURN TO NORMAL
III
NORMAL ENERGETIC TREAT MENT PREVENT PATIENT FROM GOING INTO STAGE
IV
STAGE OF MEDULLARY COMPRESSION
V
BEGINING MEDULLARY EDEMA
VI
ABSOLUTE FIXED
MEDULLARY EDEMA
PULSE RATE
BLOOD PRESSURE
CEREBRO SPINAL FLUID PRESSURE MANOMETER
NORMAL
BASE LINE
DEATH
INFUSION IF NECESSARY
THREE BY ESTIMATING THE PATHOLOGY AND GENERAL CONDITION PLAN TREAT-MENT
REPEATED SPINAL PUNCTURE AND DEHYDRATION DECOMPRESSION IF THERE IS NO IMPROVE MENT
REPEATED SPINAL TAP DEHYDRATION CISTERNA TAP DECOMPRESSION
PATIENT IS MORIBOND
TREAT MENT OF NO AVAIL

Fig. 1.

——— Blood Pressure
- - - - - - Cerebrospinal Fluid Pressure
— — — Pulse Rate

without passing through the intervening stages. When once he reaches Stage 6 the patient rapidly succumbs.

Stage 2. Here we see a return to normal as regards pulse rate and blood pressure, the two criteria denoting recovery from shock. This stage represents the favorable results following the therapeutic measures used to overcome the condition of shock seen in Stage 1.

Stage 3. This stage we believe to be the most important, the pulse rate and blood pressure having returned to normal. In slight craniocerebral injuries the patient remains in this stage for a short time and rapidly recovers. In the more severe cases he passes through Stages 1 and 2, the blood pressure and pulse rate returning to normal in Stage 3. Complete physical examination, including roentgenograms of skull, neurological examination and any special examination deemed necessary, is followed by active therapy. Spinal puncture in this stage is of utmost importance these factors are the indications for treatment. This stage is represented by normal pulse rate and blood pressure; however, the patient may or may not be unconscious. The spinal fluid is usually under increased tension. We recently had an example of this stage, when 2 patients were admitted to the hospital twenty-four hours after receiving head injuries. Both were unconscious; blood pressure and pulse rate normal; the cerebral spinal fluid, however, showed a pressure of 30 mm. of mercury and was bloody, demonstrating the unreliability of pulse rate and blood pressure in determining the increase of intracranial pressure, and showing also the importance of estimating the cerebrospinal fluid pressure by the manometer. Both patients entirely recovered after conservative treatment, having never passed beyond Stage 3.

We believe and teach that patients should never pass beyond Stage 3, if prevention of this is possible. In other words, this is the period for energetic

treatment designed to promote the return of the patient to normal.

Stage 4. This is the stage of medullary compression and is characterized by slowing of pulse rate and rise of blood pressure. In this stage intracranial pressure has increased rapidly and has produced medullary compression. This possibility should be anticipated in Stage 3 and its occurrence prevented, if possible. If there is no improvement in twenty-four to forty-eight hours under conservative treatment, i.e., dehydration by the use of intravenous glucose and magnesium sulphate by mouth or rectum, subtemporal decompression is indicated. We believe that in this stage it is not advisable to wait too long before resorting to operative interference if conservative therapy fails.

Stage 5. In this stage the patient rapidly passes from beginning medullary edema to absolute fixed medullary edema. In incipient medullary edema, as shown by a fall in blood pressure and a rise in pulse rate, energetic treatment may turn the tide in favor of recovery; however, the margin of safety between early medullary edema and complete medullary edema is very small. We believe that when a patient has been seen early and treated adequately he should never reach this stage. Here our entire armamentarium should be used; dehydration, spinal puncture, cisterna puncture and decompression may restore the normal condition.

Stage 6. In Stage 6 the patient shows all the phenomena of a fixed medullary edema. Treatment in this stage is of no avail.

The chart which the author has devised for purposes of visualizing the pathology of head injuries and their treatment is shown in Figure 1. It represents a graphic picture of the phenomena taking place in acute traumatic craniocerebral injuries.

RIVANOL DEXTROSE

A NEW ANTISEPTIC FOR APPLICATION TO THE MUCOUS SURFACES OF THE URINARY TRACT*

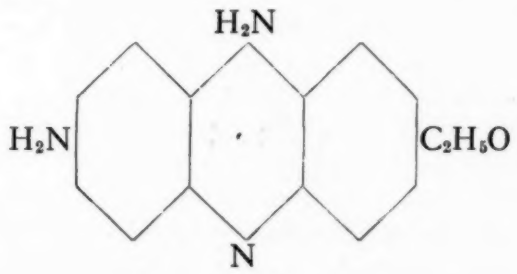
THOMAS CLEMENT HILL, B.S., M.D.

NEW YORK

THE chemical compound rivanol was first introduced into this country in 1921 from Germany. At this time the therapeutic results were satisfactory in the hands of the few who tried it, but the irritating qualities were so great that its use was abandoned. About one year ago its German originators succeeded in eliminating its irritating qualities by combining dextrose, 2 parts, to 1 part of rivanol lactate, the original preparation.

Rivanol dextrose was worked out by Morgenroth along the same chemotherapeutic principles as conceived by Ehrlich, and implied a systematic search for chemical substances with a strong affinity for parasites and a weak affinity, or possibly none, for cells of the host.

Rivanol together with acriflavine belongs to the acridine group but is superior in that it has less toxicity and great antiseptic powers. The construction of this dye was given as



and the soluble hydrochlorate was used.

Morganroth's description is as follows: a light yellow fine crystalline powder, soluble in approximately 260 parts of water at 25°C. and 28 parts of hot water. The solutions are yellow and fluorescent but darken when exposed to light for several days. The aqueous solution is stable to boiling. The reaction to litmus is neutral. The dextrose is added to the rivanol lactate to increase solubility and to prevent irritation. Our clinical results obtained in the treatment of acute gonorrhea indicate that the dextrose also activates the rivanol.

Morgenroth determined the antiseptic power* of rivanol in living tissue. A dilution of 1:40,000 sterilized an experimental streptococcus which showed phlegmon in the subcutaneous tissue of the mouse within twenty-four hours. In the test-tube streptococci were destroyed in the presence of serum by concentration of 1:100,000, this giving the favorable antiseptic index of 40,000/100,000 = 1/2.5. In staphyloc cic infections the index was greater than 1, the ideal index being 1.

The lethal dose for the rabbit is 100 mg. for each kilogram of body weight if given subcutaneously, and 50 mg. if given intravenously. This slight toxicity permits the use of large amounts of the diluted concentration in man. Rivanol has proved to have a strong affinity toward the cocci group and has been used clinically as a tissue antiseptic in such cases as localized pyogenic abscesses, pleural empyema, peritonitis and joint infections. In this article we are most concerned with its use as an antiseptic on the mucous membrane of the urinary tract.

In studying the therapeutic use of this dye, we have made a study of the comparative toxicity of neutral acriflavine (trypaflavine), rivanol lactate and rivanol lactate dextrose (1 part rivanol, 2 parts

* The absolute antiseptic index is a ratio between the antiseptic concentration of a drug in vivo and in vitro, the ideal index being 1/1, when the action of the drug in the living tissue is not inhibited in comparison with that in the test-tube.

* Read before Section of Genito-Urinary Surgery, New York Academy of Medicine, February 15, 1928.

dextrose) when injected intravenously into the albino rat.

Healthy, non-pregnant albino rats weighing 90 to 140 gm. were employed. The rats were obtained from one source and were kept on a constant well-balanced diet (a mixture composed of whole wheat meal, 53.5 per cent; corn meal, 10 per cent; skim-milk powder, 33 per cent; calcium carbonate, 0.5 per cent; sodium chloride, 1 per cent, and cod-liver oil, 2 per cent) for two weeks before the tests. They were fasted for from sixteen to twenty hours immediately before the administration of the drugs, when they were weighed and the doses administered per kilogram body weight. The solutions of the drugs were prepared with freshly distilled water and were administered within two hours from the time of preparation. A 1 per cent solution of the acridine dye was employed. The injections were made into the saphenous vein at the rate of 0.1 c.c. solution every fifteen seconds. All animals that lived were observed for six days when they were killed and carefully necropsied.

Table I

TOXICITY OF NEUTRAL ACRIFLAVINE 1 PER CENT SOLUTION

Rats Employed	Killed	Per Cent	Mg. Solution per Kg. Body Weight
3	0		50
3	0		60
3	2	67	70

Two of the animals that received 60 mg. per kilogram and all three rats that received 70 mg. per kilogram developed convulsions at the end of injection.

The intravenous maximal tolerated dose and fatal dose of neutral acriflavine is 70 mg. per kilogram. Death occurred in asphyxial convulsions within five minutes after injection. At necropsy the skin, subcutaneous tissues (including muscles) and viscera except liver and spleen were heavily stained yellow. The lungs were distended and congested and contained petechiae. Blood taken directly from the heart showed extensive agglutination of erythrocytes and a yellow precipitate several times the size of an erythrocyte.

The animals that lived were apparently normal within a few minutes after injection. They gained in weight during the observation period (six days). They were then killed. The necropsies were negative.

Table II

TOXICITY OF RIVANOL LACTATE 1 PER CENT SOLUTION

Rats Employed	Killed	Per Cent	Mg. Solution per Kg. Body Weight
3	0		40
3	0		60
3	0		80
3	1	33	100
3	3	100	120

Two of the animals that received 60 mg. per kilogram and all of the animals receiving 80 mg., 100 mg. and 120 mg. per kilogram developed asphyxial convulsions at the end of the injection.

The intravenous maximal tolerated dose of rivanol lactate is 100 mg. per kilogram and the fatal dose is 120 mg. The death at 100 mg. per kilogram occurred within twenty-four hours and the deaths at 120 mg. per kilogram occurred immediately in asphyxial convulsions at the end of the injection. The skin, subcutaneous tissues (not muscles) and viscera except spleen were faintly stained yellow. The lungs were distended and congested. Blood taken directly from the heart showed agglutination of erythrocytes, possibly some hemolysis, and a yellow precipitate.

The animals that lived were apparently normal within twenty-four hours. All gained in weight during the observation period (six days) except those receiving 100 mg. per kilogram. They were then killed and necropsied. The necropsies were negative except for the animals that received 100 mg. per kilogram. The kid-

neys of these animals showed definite nephrosis and the liver of one was swollen, mottled greyish yellow, and friable.

Table III

TOXICITY OF RIVANOL LACTATE-DEXTROSE MIXTURE 3 PER CENT SOLUTION

(RIVANOL LACTATE, 1 PART; DEXTROSE, 2 PARTS)

Rats Employed	Killed	Per Cent	Mg. Solution per Kg. Body Weight
3	0		40
3	0		60
3	0		80
3	2	67	100

Two of the animals receiving 60 mg. per kilogram and all of the animals receiving 80 mg. and 100 mg. per kilogram developed asphyxial convulsions at the end of the injection.

The intravenous maximal tolerated dose and fatal dose of rivanol lactate dextrose mixture was based upon the amount of rivanol lactate injected in 100 mg. per kilogram. Death occurred in asphyxial convulsions immediately after injection. Necropsies are the same as after rivanol lactate alone.

The animals that lived were apparently normal within twenty-four hours. All gained in weight during the observation period (six days) except the animal receiving 100 mg. per kilogram. At necropsy one of the rats receiving 80 mg. per kilogram showed slight nephrosis and the rat that received 100 mg. per kilogram showed moderate nephrosis. The necropsies were otherwise negative.

The fatal dose of neutral acriflavine is 70 mg. per kilogram when injected intravenously into the albino rat as a 1 per cent water solution. The fatal dose of rivanol lactate is 120 mg. per kilogram and the fatal dose of the rivanol lactate dextrose mixture based upon the amount of rivanol lactate injected is 100 mg.

Neutral acriflavine and rivanol lactate agglutinate the erythrocytes and form yellow precipitates when injected intravenously into the albino rat as a 1 per cent water solution. The addition of 2 per cent glucose to the rivanol lactate solution does not influence this agglutination or precipitation.

Although our work with this dye is recent and rather limited, we feel that the results obtained over the silver salts and other acridene derivatives are sufficiently creditable to warrant a recital of our experience.

Rivanol dextrose solution was used for irrigations of urethra and bladder in concentration of 1:5000 and, if well tolerated, was increased to 1:3000. For acute infections of the urethra, 1:1000 solutions were used as an instillation.

Table IV will give some idea of the results of treatment which we have been following in a small series of cases over a limited time:

The diseases treated were confined to infections of the urethra and bladder, being gonorrheal, mixed infectious or tubercular in type. There were 20 cases of acute gonorrheal anterior urethritis. These were treated by daily anterior instillations of rivanol dextrose 1:1000. Fifteen, or 75 per cent, were pronounced cured after five weeks' treatment, that being the period of time considered sufficient to insure a cure. In general, the gonococci disappeared within ten days, the variation being from four to eighteen days.

The purulent discharge disappeared in 1 case after the first injection, in 2 after the third and in 3 after the fourth. In 6 other cases purulent discharge had disappeared by the tenth day. The 3 other cases had a recurrent discharge from three to four weeks after treatment started but these were scanty and noticeable usually in the morning and all were well at the end of five weeks. The other 5 cases did not yield well to treatment. One case showed gonococci organism for four months and developed prostatic and posterior urethral complications. The 4 remaining cases developed posterior urethritis with persistent discharges, but repeated examinations of the

TABLE IV

CHART OF CASES TREATED

Diagnosis	No. of Cases	Duration of Regular Treatment	Successful Treatment		Av. Disappearance Specific Organisms	Variation of Disappearance Specific Organisms	Disappearance of Purulent Discharge					Variation of Disappearance of Purulent Discharge
			Cases	Per Cent			1 day	3 days	4 days	10 days	10–28 days	
							1 case	2 cases	3 cases	6 cases	3 cases	
Acute anterior gonorrheal urethritis.....	20	5 weeks	15	75	10 days	4–18 days						1–28 days
Post-urethritis mixed infection.....	5	3–5 months	3	60	Complications: 2 chronic prostatitis. 1 chronic prostatitis and cystitis							
Tuberculous cystitis...	3		Improved, symptoms greatly relieved, 3 cases, capacity of bladder increased									
Chronic prostatitis....	30		Improved with massage and irrigation with rivanol dextrose									
Pyelitis.....	10	6 weeks	8 cases showed definite improvement in symptoms									

smears were negative for gonococci after five weeks' treatment. These uncured cases were evidently of a mixed infectious origin and seemed to respond best to a combination therapy of rivanol and silver salts used as irrigations first of the bladder then in a more concentrated solution as anterior and posterior instillations.

Three cases of tuberculous cystitis were treated at this time, using rivanol dextrose, 1:5000, as an irrigation. This proved especially effective as compared to the results of previous treatments with acriflavine and the silver salts solutions. It has a great analgesic, antiphlogistic and secretion-diminishing action in severe forms of cystitis. The anodyne action was especially remarkable in these cases and under the influence of rivanol dextrose the inflammatory catarrhal manifestations improved rapidly, the sclerotic changes became diminished and the capacity of the bladder greatly increased. We have also been using rivanol dextrose as a posterior instillation in treatment of prostatic infections, but are unable to give any valuable information as to the results in these cases, other than to say the results are equal to those with any other preparation we use.

Numerous cases of cystitis of mixed infectious origin responded to treatment in a similar manner as those cases of cystitis mentioned above, the urine clearing up rapidly and the symptoms usually being relieved.

Recently we have begun a routine irrigation of our pyelitis cases with rivanol dextrose, 1:2000, and have had definite improvement in most cases. The time during which we have been using this treatment has not been sufficiently long to form accurate conclusions.

SUMMARY

1. The chief value of rivanol dextrose in infections of the urinary tract as seen in clinical observations, aside from the possible sterilizing properties by a non-toxic and non-caustic agent, is the building up of an antiseptic wall between the focus and the general circulation by means of circular infiltrations around the progressing infection.

2. Rivonal dextrose has marked efficiency in the treatment of acute gonorrheal urethritis with its accompanying analgesic action and relief of symptoms.

3. Lack of toxicity permits the use of large amounts of the diluted solution.

4. Transparent and stainless properties are valuable.

FAT IN TRAUMATIC EFFUSIONS OF THE KNEE JOINT*

DAVID H. KLING, M.D.

LOS ANGELES

DURING a study of effusions the presence of fat in hemorrhagic fluids aspirated from the knee joint was observed. This condition was new to the writer and no account of it was found in the literature. Therefore, an investigation was made of the appearance, the source, and the significance of fat in traumatic effusions of the knee joint, the results of which are submitted in this paper.

APPEARANCE

Fat was found in bloody knee-joint fluids only. When present in considerable amount it can be discovered without difficulty. If the fluid is poured into a dish, the fat droplets will be seen on the surface, much like fat "eyes" in bouillon. If a bottle containing the fluid is shaken, fat will stick to the walls and form a turbid film. Small amounts of fat and fat particles which are solid at room temperature (palmitin and stearin) can be recognized by centrifugalizing the fluid for five to ten minutes. The centrifuge tube offers a very characteristic appearance (Fig. 1). The fluid is separated in three or four layers. Blood corpuscles form the sediment at the bottom of the tube.

The second layer is liquid and contains the serum which is yellow or reddish colored, according to the degree of hemolysis present. The fluid may be either clear or turbid when flocculi of mucus are floating or fat droplets are emulsified in the serum. This layer is the largest and makes up from one-half to three-fourths of the contents of the test-tube.

The third layer is solid, whitish colored, and consists of the neutral fats, palmitin and stearin, which have a melting point of 66°C. and 69°C. respectively. They form a skin at room temperature, like cream on milk.

The fourth layer is liquid yellowish, and consists of olein which has a melting point of −4°C. It is variable in thickness, depending on the amount of olein. Considerable amounts of olein form a distinct fourth layer of oily fluid. In tube B (Fig. 1), the liquid fat is about ¾ in. thick and nearly twice the size of the third, solid layer. In effusions which contain only small amounts of olein the fluid will be separated in three layers only, as in tube A.

The line of separation between blood corpuscles and serum is very sharp and is well marked between the other layers.

The fats in fresh fluids give the physical and chemical reactions of neutral fats (esters).

If a centrifuge is not available, the sedimentation and separation in the different layers can be observed by simply placing the aspirated fluid in a narrow test-tube from twelve to twenty-four hours.

SOURCE OF THE FAT

1. The pyramidal space (Fig. 2) between the patella (above), the tibia (below) and the patellar ligament (front) is filled by a thick pad of fat. Two triangular fringes, the plicae alares, project into the joint cavity from the medial and lateral margins of the patella. They converge in the middle line and form a narrow band of fat, the plica synovialis patellaris, which crosses the joint cavity and is attached to the intracondyloidal notch of the femur in the neighborhood of the anterior crucial ligament. The fat pad and its fringes are within the joint capsule but outside of the

*From the Laboratory of the Golden State Hospital. Submitted for publication June 19, 1928.

synovial membrane. The latter reflects and folds over the plicae alares and forms a sheat around the plica synovialis.

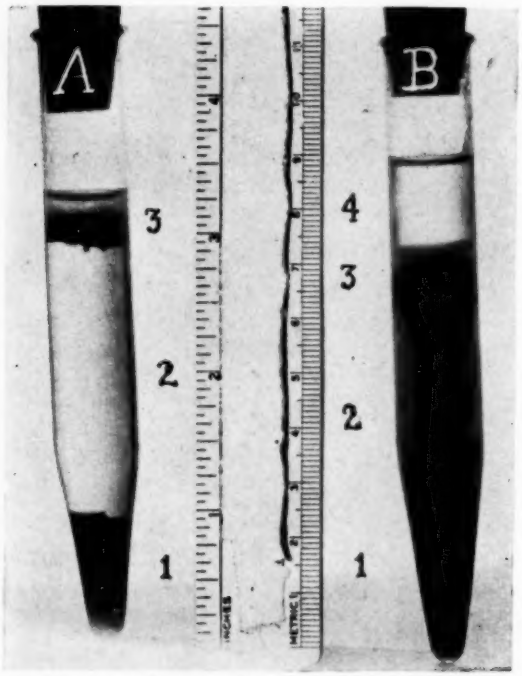


Fig. 1. Two specimens of hemorrhagic fluid from the knee joint, containing fat. A. Centrifugalized in three layers, serum clear, small amount of olein. Separated into four layers, large amount of olein: (1) blood corpuscles; (2) serum; (3) solid fat (palmitin and stearin), and (4) liquid fat (olein).

2. A variable amount of fat is enclosed in the synovial sheath of the crucial ligaments, especially at the point of their attachment to the bones.

3. The red bone marrow of the epiphysis of femur and tibia contains, besides blood and giant cells, some fat deposits.

Each of the three fat depots is, therefore, in close approximation to, but outside of, the central cavity of the knee joint. When fat is present in this cavity and demonstrated in the aspirated fluid, we are entitled to assume that the synovial membrane has been torn or the articular surface of the bones fractured and the marrow opened.

Other deposits in the region of the knee joint do not contribute fat to the aspirated fluid.

There is some fat around the suprapatellar bursa but it is outside of the capsule and well protected against injuries. The writer has not observed to date fat in fluids from this bursa, but in case it should occur, we must make sure that it did not originate in the central cavity and reach the bursa through a communication.

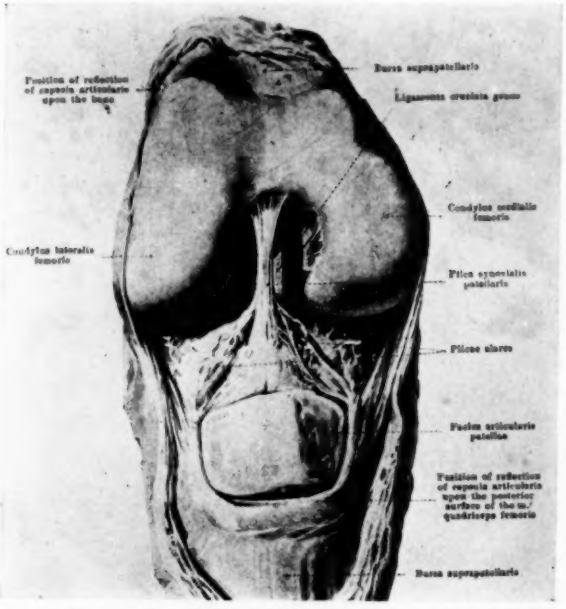


Fig. 2. Fat pads of the knee joint. (*From Spaltebolz's Hand Atlas of Human Anatomy.*)

The prepatellar and infrapatellar bursae are separated from the knee joint. There are holes in the posterior wall of the capsule at the entrance of the blood vessels which are filled with fat particles. This wall, however, is not exposed to the common injuries and fluid accumulating in the posterior pouch can not be aspirated from the front.

SIGNIFICANCE

Diagnostic Consideration. In a series of 15 cases of injuries to the knee with hemorrhagic effusions fat was found in 6 (39.6 per cent) and was absent in 9 (60.4 per cent) of the fluids. The diagnosis in the latter 9 cases was: suprapatellar bursitis, 2; prepatellar bursitis, 5; synovitis, 2 cases. Roentgenograms were negative and symptoms of injury of the internal ligaments and cartilages of the joint were absent in these cases. The centrifugalized fluids were separated in two layers; at the bottom a sediment of blood corpuscles, followed by a more or less hemolytic, clear or turbid serum.

The clinical diagnosis of the 6 cases with fat present in the effusion was:

1. Rupture of the internal semilunar cartilage.
2. Rupture of the internal semilunar cartilage and anterior crucial ligament.
3. Detachment of internal semilunar cartilage.
4. Infraction of external spine of tibia, rupture of posterior crucial ligament.
5. Infraction of intercondyloid notch of the femur, rupture of anterior crucial ligament and mucous ligament.
6. Infraction of medial spine of tibia and medial condyle of femur and rupture of both crucial and external collateral ligaments.

All 6 cases suffered an injury of the internal ligaments or cartilages of the knee joint. Three cases were complicated by fractures of the articular surfaces of femur and tibia. The aspirated fluid centrifuged in three or four layers as described above.

The importance of the finding of fat in knee fluids is made evident by a comparison of this group and the first series of cases where blood alone was aspirated.

Bleeding is not symptomatic for a severe or extensive injury of the knee joint and it does not indicate the site of the damage in the synovial membrane. Different bursae were involved in the first 9 cases.

Fat, on the other hand, is indicative of a severe injury where the synovial membrane of the central cavity has been torn and the interjoint ligaments and cartilages damaged, eventually combining with a fracture of the articular surfaces. The latter point will be decided by roentgenography. The writer found the amount of olein (fourth layer) relatively high in the cases complicated by fractures.

A consideration of the blood supply of the knee joint will explain the entirely different significance of the finding of blood and fat in traumatic effusions of this part. A dense net of vessels (rete articulare genu) supplies all pouches, folds and bursae. Hence, a small tear at any point of the synovial membrane causes bleeding, the amount of which depends on the condition of the vessels as much as the size of the tear. Heart and kidney conditions, hypertension, arteriosclerosis, lues and diabetes make the vessels vulnerable. Hemorrhage is, finally, not exclusive proof of a previous injury. Bloody effusion in the knee joint can be an early sign of hemophilia. Also sarcoma of the synovia and capsule can produce an hemarthrosis. These affections may be rare but they are of diagnostic interest and importance in deciding liability in compensation cases. But the fat depots which are exposed to trauma are located around the central pouch of the knee joint and in the close neighborhood of the interjoint structures. Fat is not known to be affected by the mentioned conditions.

Only a severe trauma which produces extensive damage to the synovial membrane of the central cavity and to the interjoint structures or articular surfaces of the knee joint is able to tear particles of fat from the depots and deposit them in the joint cavity.

While the differential symptomatology of knee injuries has been worked out thoroughly, it is still a matter of considerable difficulty to recognize the nature and extent of injury in the acute stage when the joint is very painful and distended by a large amount of fluid. Examination of the fluid for the presence of fat will supply valuable information and help solve diagnostic and prognostic problems.

Pathogenic Consideration. Blood is slowly resolved and absorbed in serous cavities, but we know nothing of the way in which fat could be eliminated there. Enzymes which accelerate hydrolysis of fatty esters (esterases) were found in all tissues but only traces of true lipases were discovered outside of the pancreas and intestines. Fat, especially the solid components, palmitin and stearin, may precipitate in the joint cavity and in consequence undergo organization. This explains the formation of loose bodies, consisting of fibrous fat after injuries. It induces, therefore, a chronic pathological condition

which prevents the restitution of the normal function of the knee joint.

Therapeutic Consideration. Traumatic effusions were formerly not touched and the knee joint was immobilized for a considerable time. Recently the authors on this subject recommend early aspiration, repeated in case effusion recurs. This procedure together with early mobilization prevents atrophy, restores the tonus of the capsule and removes clots and fibrin which tend to precipitate. This method of treatment is fully supported by the discovery of fat in severe injuries and the possibility of organization of fat particles with formation of loose bodies in the joint cavity. It is proved that the aspiration is not connected with any danger of infection when the ordinary aseptic precautions are taken, and is harmless even in septic and tuberculous effusions.

As an illustration, two histories will be abstracted briefly, the first a case of internal derangement alone, the second combined with an interjoint fracture.

CASE REPORTS

Case 1. W. L., white, aged thirty-nine, butcher, slipped and fell on a piece of ice in the refrigerator on December 15, 1927. He hurt his left knee but was able to finish his work and walked home, a distance of several blocks. In the evening he notcied that the knee was markedly swollen and very painful. Next morning he was transferred to the hospital. There was no previous injury or inflammation of this joint.

Examination found the knee joint very much swollen and extended. All movements were painful; the knee was locked in partial flexion. There was marked tenderness over the inner knee joint. The roentgenogram revealed no injury to the bones.

Diagnosis was rupture or dislocation of internal semilunar cartilage.

Forty cubic centimeters of bloody fluid were aspirated. Upon being centrifugalized it separated into three layers. At the bottom was a layer of blood corpuscles ½ in. thick, above which was a layer of cloudy serum 4 in. thick. The cloudiness of this layer was due to suspended fat. On the top was a thin skin of solid neutral fat.

After aspiration the knee was extended fully. A snapping sound was heard during this manipulation.

Case 11. P. H., white, aged twenty-four. While unloading iron beams on February 1, 1928, the patient was struck by a falling pile. The right knee was injured so that he could not raise it. He was transferred to the hospital.

Examination showed the knee joint and the adjacent tissues swollen. All movements were painful. Superficial abrasions were found on the anterior surface of the knee. The roentgenogram revealed infraction of the internal condyle opening in the knee cavity with slight displacement of the fragments, and an infraction of the inner spine of the tibia and abnormally wide separation between the outer condyle of the femur and the corresponding fossa tibiae.

Diagnosis was intercondyloidal fracture of the femur and fracture of the medial spine of the tibia, with rupture of both crucial and external collateral ligaments.

Fifty cubic centimeters of hemorrhagic fluid were aspirated. Numerous fat droplets floated on the surface of the fluid. In the centrifuge the specimen separated into four layers. At the bottom were blood cells to a depth of ⅛ in. Next there was a layer of clear reddish serum 2 in. thick, covered by a skin of white solid fat ⅛ in. in thickness. The top layer was liquid, lemon yellow in color, about ¾ in. thick, and was composed of olein. Physical and chemical reactions were positive for neutral fat.

SUMMARY

1. Fat was observed in traumatic hemorrhagic effusions of the knee joint.

2. Fat is conveniently discovered by centrifugalizing the aspirated fluid. It accumulates on the surface and causes separation of the fluid into three or four layers.

3. The interjoint fat depots and the bone marrow of the epiphyses are the source of fat in the aspirated fluid.

4. The fat indicates an injury of the interjoint structures (crucial ligaments and semilunar cartilages) alone or combined with fracture of the articular surfaces.

5. Fat particles torn and deposited in the joint cavity may be organized and form loose bodies.

6. Early aspiration is indicated.

RUDIMENTARY HORN PREGNANCY*

WITH REPORT OF CASE

ABRAHAM KOPLOWITZ, M.D.

BROOKLYN

PERHAPS no field of congenital anomalies presents such a confusion of terminology as that referring to anomalies of growth of the female generative organs. Frequently different names have similar meanings. For instance, the term uterus unicornus, while anatomically restricted to asymmetry of development with absence of Fallopian tubes, is also applied to designate those cases showing a rudimentary horn and tube.

Embryologically, the uterus, tubes and vagina are formed by the proper development and proper fusion of the two Müllerian ducts, which fusion takes place from below upwards. Lack of fusion, resulting in an anomaly varying in degree from the uterus arcuatus to the uterus didelphis where there exist two separate uteri, cervici and vaginae, accounts for most of the anomalies. Rudimentary development of one of the ducts accounts for the rest.

We are concerned in this paper with that failure of development of one of the ducts resulting in a rudimentary horn attached to an apparently normal uterus, so far as appearance shows, or a uterus apparently onesided (unicornis), evidently developed of one Müllerian duct though with a complete cervix and vagina.

According to Aschoff's classification of asymmetric hypoplasias and aplasias of the uterus, this is called uterus bicornis cum acornu rudimentaria. That is, one duct is fully developed, the other defective in vagina and cervix and situated as a rudiment on the other half of the corpus uteri. Eden and Lockeyer, for the same condition, use the term uterus unicornis with rudimentary horn. De Lee's term, terus bicornis unolatere rudimentaria, seems perhaps most appropriate because most expressive.

The rudimentary horn is usually situated on the right side, though no reason seems to be known for this. Most frequently a well-developed tube and ovary are attached to it and the round ligament is usually attached to this horn close to the body of the uterus. This connection of the rudiment with the rest of the corpus is usually by a pedicle to the upper angle or towards the middle of the body of the well-developed uterus. This band gives a direct communication with the rest of the uterine cavity in about 20 per cent of the cases on record, though Humpstone, quoting Brooke Wells, claims that a much greater percentage would be found if a more careful search were made, asserting that a microscopical study of cross sections of the pedicle might reveal a sufficiently large canal to permit a passage for a spermatozoon.

The rudimentary horn has a mucosa identical with the endometrium and is continuous with the tube on that side. It may cause hematometra and hematosalpinx and may become the seat of pregnancy. This may occur through the opening in the pedicle, if one exists, or by transperitoneal migration of the spermatozoon if the ovum is from its own side, or of the impregnated ovum if from the opposite side.

From the time of Mauriceau and Vassal in 1669, who reported the first case, De Lee states that there were 100 cases reported up to 1916. Wardlaw and Smith have collected some more to 1922. Quain in 1913 claimed to have collected 150 cases from the literature, though he admits that the incompleteness of some of the reports and general confusion of terms would make that number much less. Thus he quotes Kehrer in 1900 reporting 82 cases, and Cohen, in 1909, 50 more, though many were

* Submitted for publication Aug. 22, 1928.

questionable on account of confusing nomenclature.

Guillaume in 1911 tabulated 145 cases, not claiming to have exhausted the literature. However, a study of his work tends to show that several cases included were really uterus bicornis or uterus subseptus. It is quite remarkable how few have been reported by American surgeons, the first being by Paul Munde, in 1890.

Of all the cases reported, about 30 are said to have gone to full term. But a closer study of these advanced cases have convinced Quain that this number is much too high. Several authors have described a dead fetus which had died at "term" while as a matter of fact either the history would tend to show that the fetus had died a month or more before term or the description of the specimen would fail to prove a development to full term.

The conclusions as to the length of the pregnancy must rest very largely on the examination of the fetus, the actual size being of less importance than the development of special tissues and organs. Quain's case unquestionably lived to term, as shown not only by the history but by the physical examination of the fetus. Skiagraphs showed advanced bone development, especially the centers of ossification of the lower epiphyses of the femurs.

Scott and Forman reported one of the most interesting cases on record where 2 full-term fetuses were retained in a rudimentary horn for twenty years. The pregnancy from which this resulted occurred after two living children were born and was followed by the birth of another living child.

Humpstone in 1920 reported 2 cases, 1 of about four months, which gave a typical history and findings of ectopic pregnancy which had ruptured, and a second, which was admitted as a full-term pregnancy in eclampsia, the peculiar anomaly being discovered at the cesarian operation which followed. It is interesting to note that he had the same experience as we did in the first case the writer reported, that is, in attempting to perform a vaginal hysterotomy, the surgeon, Dr. Harvey Matthews, unexpectedly found himself in the peritoneal cavity. According to Humpstone these cases divide themselves in two classes: those in which pregnancy terminates in four to five months by rupture and hemorrhage (about 90 per cent of the whole) and those in which there is sufficient musculature in the rudimentary horn to allow enough hypertrophy for the condition to go to, or near, full term, when an attempt at labor sets in.

Even then, rupture may take place, with escape of the fetus into the abdomen, but the hemorrhage is not as severe as that occurring in a tubal rupture, because the muscular cyst walls retract, compressing the torn blood vessels and firmly grasping the contained placenta after the escape of the fetus. When rupture does not occur, labor soon ceases, death of the ovum takes place and absorption of fluids quickly reduces the mass in size.

A clinical diagnosis of pregnancy in such a horn has been made in but few instances before operation. Among these were those of Draghiesco (quoted by Cohen), Hoene, Knauer and Wehle (quoted by Guillaume), and Lindner in 1904. Since 1922 the only cases reported in German and English literature were those by Kriwsky, of a nearly full-term pregnancy, and by D'Arcy of a full term, both in 1925. A fair review of American literature disclosed only 12 cases altogether, with 3 full-term cases among them: Munde in 1890, Wells in 1900, Webster in 1904, Frankenthal in 1910, Quain (full term) in 1913, Schwartz in 1915, Scott and Forman (full term) 1916, Humpstone in 1920 (one full term), and my own in 1928 (eight months).

These cases are by no means all the cases met with, as a good many are not reported, for instance, in a discussion of Webster's case, Emil Reis stated that he had operated on 2 such cases that he had not reported.

CASE REPORT

Mrs. S. K.,[1] aged thirty-six, was admitted to the hospital on November 6, 1927, with the following history.

[1] Case No. 25706. B. E. N. Y. Hospital.

She was of a robust type and had always been well. She had had eight pregnancies which were attended by a midwife. They were uneventful; each terminated spontaneously and in normal deliveries. There were no periods of relative sterility and there were no abortions, spontaneous or otherwise. Her menses began at the age of thirteen and were regular, of a twenty-eight-day type, bleeding for three to four days, and unassociated with pain, excessive or intermenstrual bleedings. Her last regular period was three months prior to admission to the hospital.

During this period of amenorrhea she suspected that she might be pregnant, although she felt perfectly well. On the day of admission to the hospital at about 4 P.M. she was seized suddenly with sharp lancinating pains in the lower abdomen, marked weakness and fainting spells. Two hours later she began to bleed per vagina. The weakness was progressive, becoming extreme, but the pain did not abate. Dr. Krieger, who was called to see her, made the diagnosis of ruptured ectopic pregnancy and rushed her to the hospital.

The physical examination on admission revealed the following: Temperature 97°F. Pulse rate 60. Respiration 18. Blood pressure 78/60. The abdomen was distended and tender all over, especially over both lower quadrants. There were no masses palpable and there was an absence of peritoneal rebound. Vaginally she showed a fair parous introitus, small closed cervix, and the uterus could be felt by the examining finger beyond the cervix but could not be completely made out. It did not give the impression of being enlarged. Fornices and cul-de-sac were extremely tender, but there was no evident fluctuation.

The laboratory report was as follows: Blood: hemoglobin 58 per cent, R.B.C. 2,800,000, W.B.C. 11,000 with a differential of 70 per cent neutrophiles, and 30 per cent lymphocytes.

Diagnosis of ruptured ectopic, pregnancy, probably interstitial in type, was made.

With this history and these findings operation was decided upon and performed immediately. On opening the abdomen and after inspection, the following were noted: The uterus was normal in size and centrally located. To the right of the uterus and attached to the right cornua by a musculo-fibrous band was a structure which appeared like another uterus with a rent in it about 5 cm. in length, from which placenta and cord were protruding. The right tube and ovary were attached to this structure and the left tube and ovary, normal in every way, were attached to the uterus first described. Free blood and many blood clots, together with a fetus the size of a two and a half to three months' gestation attached to the cord, were found free in the abdomen. The structure containing the rent and the right tube and ovary were removed. Three hundred cubic centimeters of a 10 per cent solution of glucose were left in the abdomen and 450 c.c. of a 10 per cent solution were given intravenously.

The patient reacted completely and did well for eight days, when the temperature rose suddenly to 103.6°F. She complained of pain in the left side and the spleen could be palpated. Diagnosis was made of splenic infarct. She recovered completely and left the hospital eight days later (sixteen days postoperative) in excellent condition.

The specimen removed was examined by Dr. R. Frank, who reported the following, and made the accompanying drawings based on the specimen and the report of findings at operation:

The specimen consists of one half of a uterus bicornis, the horn of the uterus being 6 cm. long, 5.5 cm. wide and 2.5 cm. at the base removed from the other horn. A right Fallopian tube, normal in size and appearance, occupies one side. The abdominal ostium of this tube is normal and open. The tube takes a somewhat anomalous course at its uterine end, running superficially on the posterior surface of the fundus and then penetrating into the uterine musculature, approximately 0.3 cm. from the edge.

Below this tube is a normal ovary containing only an old, well-involuted corpus luteum, evidently antedating the pregnancy by a number of weeks. The fundus in gross appearance closely resembles a normal fundus except that at the usual site at which the left adnexa should be, is a large circular defect about 2 cm. in diameter, penetrating into the interior of the uterus and occupied throughout its circumference by chorionic tissue. No evidence of left adnexal insertion can be found. The basis of attachment of this uterine horn contains no canal (this was confirmed by microscopic section of this area). An almost circular cavity occupies the inside of the uterus. This cavity is lined by endometrium and communicates with the tube (microscopically confirmed).

In addition a free piece of placental material

about 7 by 1 cm. in size was included in the specimen.

The fetus is 10 cm. long, apparently male in sex, attached to a cord 15.5 cm. in length, evidently between two and two and a half months in age.

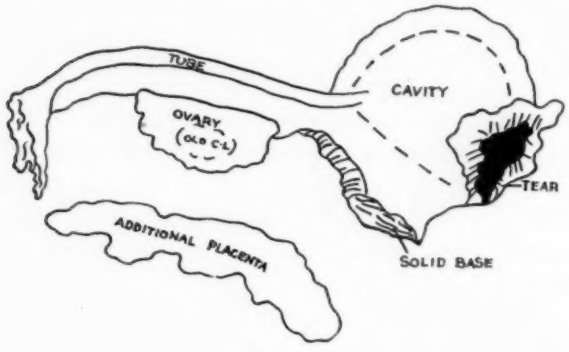


Fig. 1.

The fact that no fresh corpus luteum was found on the involved side makes it evident that fertilization had taken place through the cervix of the left, evidently pervious cavity, and that the ovum rose from the left ovary, was taken up by the right tube and implanted in the right horn, which had no cervical outlet. In the further course of the gestation, rupture of this horn took place into the abdominal cavity.

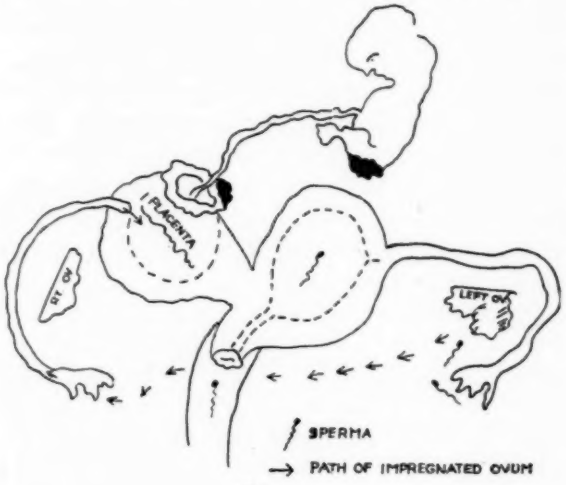


Fig. 2.

This then is a clear case of transmigration of the ovum across the peritoneal cavity.

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RENAL PELVIS OBSTRUCTION

DUE TO ABERRANT VESSELS*

W. H. McNeill, M.D.

NEW YORK

THE origin of anomalies of the renal vessels was not definitely determined until Jiedel and Bremer made some embryological investigations in 1915. Their conclusions were:

1. The anomalies of the renal artery depend on vessels present in the embryo or upon the aorta and its larger branches which develop from the mesodermal coats from which there are no late branches.

2. Certain anomalies are due to the persistence of the early renal blood supply most frequently seen in pelvic kidneys that is, the renal artery has a branch from the iliac, inferior mesenteric or the middle sacral artery or from the aorta below the middle mesenteric artery.

3. A periaortic plexus of vessels with many roots from the aorta exists, affording the opportunity for the change in position of the main aortic branches and supply of the smaller branches for possible future use.

4. The renal artery is derived from this plexus. The channel for the permanent renal artery is selected mechanically. Channels not so convenient may be utilized if the usual channel is occluded, hence the renal variations and anomalies. These changes take place while the kidney is migrating from its original embryonic pelvic position to its permanent one.

Some authorities believe that this anomaly in all its different types exists in 20 per cent of cases, but Mayo and Eisendrath report the condition to exist in about 2.5 per cent of cases.

Ekeham in 1907 was the first to direct attention to the part which the accessory vessels to the lower pole may play in the etiology of hydronephrosis.

The inferior polar artery branching from the main renal artery is a very rare condition and only one or two cases have been reported. Inferior polar arteries from the aorta are the most common and are the outstanding etiological factor in obstructing the flow of urine down the ureter, resulting in a distended pelvis of the kidney, hydronephrosis and calculus formation. The aberrant artery may also arise from the iliac and even from the middle or inferior mesenteric. Eisendrath states that lower polar arteries from the aorta, renal and iliac arteries are present in one out of every 185 kidneys.

In this series of 9 cases, 2 cases were hypernephromas:

CASE I. A man aged forty-four, complained chiefly of pain in the left costovertebral angle, radiating to the back with hematuria of one month's duration. Hematuria and pain had become progressively worse. The patient had lost weight gradually. Cystoscopy showed the left kidney full of blood and pus and of very low function. Nephrectomy was performed for hypernephroma. A large aberrant vessel entered the kidney half way between the pelvis and lower pole.

CASE II. A man aged fifty-eight, complained chiefly of blood in the urine, pain in the right flank and frequency of micturition. The symptoms had been present for ten weeks. There was general weakness. Cystoscopy showed a very low kidney function on the right side. Nephrectomy was performed for hypernephroma and aberrant vessels were found running to the anterior surface of the lower pole. These vessels resulted in some obstruction to the flow of urine but were in no way the etiological factor causing the malignancy.

There were three cases of hydronephrosis:

* Read before Section of Genito-Urinary Surgery, New York Academy of Medicine, May 16, 1928.

Case III. A man, aged fifty, entered the hospital complaining of pain in the left loin and hematuria, which he said had been present since 1898, varying in duration from one day to two weeks with intervals of no bleeding for months, nocturia three times, frequency during day. Cystoscopy revealed a large damaged left kidney. Nephrectomy was performed. The aberrant vessels in this case were imbedded in a thick fibrous band running to the anterior surface of the lower pole.

Case IV. A man aged forty, came in complaining of indigestion and mass on the right side, and dull pain radiating to the right flank. The patient was sent at first to the medical ward and finally referred to the genitourinary service for cystoscopy and pyelography. Cystoscopy revealed no urine or dye coming from the right kidney in thirty minutes. Nephrectomy was performed. In the perirenal tissue at the ureteropelvic junction was a band of tissue which at first was thought to be a double ureter but later proved to be an aberrant vessel running to the anterior surface of the kidney at the lower pole, coursing transversely to the long axis of the ureter in such a way as to form a blockage at the ureteropelvic junction. On dissection of this band of tissue, three vessels were found.

Case V. A man, aged thirty-five, entered the hospital complaining of pain in the left kidney region for the last twenty years, but decidedly worse for the past few weeks, dragging in character, aggravated by taking alcohol and acid foods. There was no hematuria, but a slight amount of frequency during the day and some nocturia. Nephrectomy was performed. A large aberrant vessel was found running to the lower pole and causing a great deal of pressure on the ureter, thereby blocking the flow of urine.

Four cases of calculi were found with some existing hydronephrosis:

Case VI. A man, aged fifty, entered the hospital complaining of pain in right lumbar region dating back one year, followed by hematuria which lasted one week and then disappeared, then returned one month ago. Pain was becoming more severe and was closely associated with the hematuria. Cystoscopic examination revealed urine filled with pus coming from the right kidney; no blue appeared in twenty minutes. Nephrectomy was performed. The vessels were found to cross posterior to the ureter just below the ureteropelvic junction. This condition was thought by the operator to be the cause of the existing hydronephrosis and calculus formation. Because of the large size of the vessel, it was felt that its ligation would cause atrophy of the lower pole of the kidney. This possibility, along with the presence of a hydronephrosis and calculus and an excellent kidney on the opposite side, indicated nephrectomy.

Case VII. A man, aged fifty-four, entered the hospital complaining of a pain in the left kidney region for the past four months, dull aching in character, and severe colicky attacks, but with no hematuria, frequency or urgency. Cystoscopic examination showed a diminished function with pus in the left kidney. Pyelotomy was performed and a calculus removed. The aberrant artery was present at the lower pole just below the ureteropelvic junction. It was also accompanied by a vein.

Case VIII. A man, aged fifty-nine, entered the hospital complaining of pain in the right kidney, loss of weight and nocturia. Symptoms had been present for the last six months, aggravated by exercise. Nocturia and frequency were marked. The patient had lost 40 to 50 lbs. Cystoscopy revealed a large amount of pus coming from the right kidney, but no blue came through in thirty minutes. Nephrectomy was performed. An artery was found running to the upper as well as to the lower pole. The operator did not believe that the vessel caused any obstruction to the ureter.

Case IX. A man, aged forty-eight, had been suffering from a pain in the left kidney region for the past year and a half with some hematuria following severe colicky attacks which radiated down the course of the left ureter. Cystoscopic examination revealed pus and blood coming from the left kidney and a diminished amount of blue. This patient also gave a history of having passed a stone with hematuria about one year ago. Pyelotomy was performed and a stone was removed from pelvis of the kidney. A large vessel running to the anterior surface of the lower pole of the left kidney was found. This vessel held the ureter down tightly and caused a marked retraction in the pelvis. This was a very excellent demonstration of the blocking that may be caused by an aberrant vessel. When the vessel was raised from the

ureter, the pelvis of the kidney readily emptied itself.

In these cases the aberrant vessels all came from the aorta. In the four calculi cases, the calculus was in the pelvis of the kidney in every case. In three of the cases (Cases I, II and VIII) the operator did not believe that the existing aberrant vessel was in any way responsible for the pathology, but in the other cases it was felt that the vessel was the mechanical factor causing the existing condition. Therefore, one may observe that vessels to the lower pole of the kidney do not always cause obstruction of the ureter, resulting in hydronephrosis and calculi formation, but in a great many instances they are responsible for the pathology found in the kidney.

In ligating these vessels, especially if they are large or many in number, one thinks of the possible atrophy of that portion of the kidney which the vessel supplies, but the actual damage caused will never be known as we are unfortunate in not being able to follow these cases in later years to the autopsy table.

DISCUSSION

Dr. Wilbolz (University of Berne, Switzerland): I was particularly interested in the cases of hydronephrosis with abnormal vessels, which I always propose to treat as long as possible in order to conserve the kidney. We can only conserve it when we have opportunity to observe the case very early, and therefore we must appeal to the general practitioner to send us these cases as early as possible. I am sure that when these vessels are cut there will always be a necrosis of the lower pole of the kidney, and later there will be a diminishing of the function and a great scar in the lower pole. When these vessels are not cut, plastic on the pelvis is all that is required, and this is not very difficult. The ureter must be cut, and then in spite of sparing the vessels the ureter can be pushed behind the vessel and there will be no more pressure. I have cases two, three, and four years after operation in which the pelvis is working very well. The function is not quite normal but it has improved and remains favorable for many years, so that we can be sure that in many of these cases we will be able to save kidneys which otherwise would have to be nephrectomized later on. For this reason I make this appeal to try more conservative work in these normal vessels with hydronephrosis.

SUBSEROUS CHOLECYSTECTOMY*

EGBERT L. MORTIMER, M.D.

BALTIMORE

TOTAL extirpation of the gall bladder by the classic method is fraught with grave risks because of the impossibility of properly closing the cystic duct. Exposure of the cystic duct in the usual operation for cholecystectomy necessitates a great deal of gauze packing, with pressure and traction on the viscera. This traction and pressure usually causes such shock to the patient that a long, stormy, uncomfortable convalescence is the result.

There have been many types of procedure suggested to overcome these defects, but none of them give the results in my hands that a modification of Doyen's subserous method for enucleation of the gall bladder does.

The late Dr. Albert Chambers and I, working on cadavers fifteen years ago, saw that the serous coat could be easily stripped from the gall bladder with the result that it could be skinned back and down to the cystic duct. By this means there was no possibility of confusing the cystic and the common ducts. The cystic duct could be ligated and left inside of the original serous sack of the gall bladder, thus giving protection to the duct and to the surrounding viscera, in preventing adhesions as no raw surfaces are exposed. In addition there was no injury to the bed of the liver so there should be no bleeding.

Doyen, in 1899 seems to have been the first operator to realize the ease of subserous enucleation of the gall bladder. In his "Surgical Therapeutics" he describes an operation for it. Since that time the value of the operation seems to have been lost. Although there have been several other methods for subserous dissection suggested during the past few years, the chief points of value of this method are:

1. The ease with which the operation can be performed.
2. The small amount of shock to the patient, resulting from lack of trauma to the bed of the liver and celiac plexus.
3. The excellent exposure of the cystic duct and the impossibility of clamping the common duct by mistake.
4. The small amount of retraction needed for the exposure of the gall bladder.
5. The few instruments needed for the operation.
6. The remarkably smooth and rapid convalescence.
7. Lack of need for drainage.

TECHNIQUE

The incision used is a modified Mayo. If the viscera in the region of the gall bladder is to be explored and the appendix is to be removed, the Mayo incision is not quite long enough. On several occasions I have found adhesions to the duodenum which on slight traction have torn. Due to the excellent exposure obtained by the longer incision, the leaking of the duodenal contents was immediately detected and easily sutured.

By placing a small round pillow under the small of the patient's back, the under surface of the liver is given better exposure. Using the modified Mayo incision enables the skin and fascia to be drawn apart better and often no retraction is necessary to view the gall bladder. However, it must be remembered that this pillow is to be removed before attempting to close the wound, as neglecting to do so will cause a great deal of unnecessary strain in closing.

The incision starts at the ensiform cartilage and goes down following the free border of the ribs to the mammary line and then goes caudad for an inch and a half, or longer, if the appendix is to be removed through the same incision.

* From the clinic of Dr. Joseph Colt Bloodgood, St. Agnes' Hospital, Baltimore.
Submitted for publication, Sept. 7, 1928.

In stout patients the fascia is divided crosswise and in lean ones lengthwise, using fingers as retractors and blunt dissectors. The oozing is checked by warm saline gauze and pressure.

The peritoneum is opened just over the gall bladder, which usually presents itself without the aid of retractors. The fundus is picked up with an Allis clamp. After careful examination, the surrounding viscera are gently packed off with a warm saline Boston pack. A circular incision is made through the serous covering of the gall bladder just below the Allis clamp, and the serous layer is separated from the gall bladder either with the handle of a scalpel or a finger covered with gauze. The serous covering is stripped back down to the junction of the cystic and the common ducts. Then placing the third finger of the right hand as close as possible to the common duct, outside of the serous covering, the contents is milked as far back towards the fundus of the gall bladder as possible. A Kelly clamp is applied to the cystic duct just below the contents. A small longitudinal incision is then made in the cystic duct just below the Kelly clamp and after walling this field off with a small warm saline pack, a blunt probe is passed down the cystic duct, into the common duct and into the duodenum, sounding for stones or obstruction. If a stone is found it is removed with the aid of various sized curettes. Making certain that there are no obstructions the cystic duct is then ligated below the incision. It is sutured and anchored and a second suture placed just a little below, using No. 2 catgut for both. The gall bladder is now amputated just below the Kelly clamp. The small pack is removed and oozing is looked for. The serous coating of the gall bladder which has been stripped off is folded back and the incised end is brought together with three stitches. The large Boston pack is then removed and the abdomen closed layer by layer with No. 2 catgut. The skin is closed with interrupted silk stitches and three silkworm gut stitches through skin and fat for reinforcement.

I have performed the operation 50 times. There have been in this series of cases no operative mortalities, no second operations for adhesions and no hernias. The following case histories indicate the small amount of shock the patient suffers.

CASE HISTORIES

Case 1. St. Agnes' Hospital No. 42,974: white, female, aged forty-eight, single.

Present illness: For over a year the patient has been suffering with pains which radiate from the region of the liver, up to the right scapula. This pain is clinching in character and there is warmness under the ribs. The pain is more severe at times than others.

Past history: Pneumonia in infancy. Small growth removed from abdomen eleven years ago. Typhoid fever eight years ago. Family history: Negative. Gastrointestinal: Considerable gas forming lately. Menses began at eleven years, regular twenty-eight day type. Pain several days before.

Physical examination: Abdomen, no palpable masses. Tenderness over the gall-bladder region. No rigidity. The cramp-like pain described above is just to the right of this tender area. No jaundice. Blood pressure, 128/85. Urine, negative. Roentgen-ray examination: Gall-bladder region not visualized.

Diagnosis: Gall stones.

Operation: July 20, 1927. Cholecystectomy. (Dr. Mortimer. Assistant, Dr. Matthey. Ether, Dr. Keech.)

Operative note: Incision was begun at ensiform cartilage and followed the free border of the ribs to the mammary line and down ½ in. Gall bladder easily located. Serous covering encircling the fundus incised. Serous covering stripped back. Cystic duct clamped, incised and probed. Gall bladder amputated and delivered intact. Stump touched with carbolic and ligated with No. 2 catgut. Serous covering drawn over the stump and fastened with No. 3 catgut sutures. Abdomen closed through and through with No. 2 catgut, skin reinforced with two silkworm gut sutures. No drains.

Patient stood the operation well and was returned to her room one hour and fifteen minutes after leaving it.

Post operative note: At the end of twelve hours the patient was slightly uncomfortable and there was a little vomiting. After twenty-four hours she was expelling flatus freely and

complained of shortness of breath. In thirty-six hours she was vomiting a small amount of bile and the shortness of breath was still present; however, she had passed a fairly comfortable night. After forty-eight hours she was fairly comfortable and was on a liquid diet. On the third day she was slightly nauseated, passed flatus, but was fairly comfortable; soft diet. Daily enemas returned dark brown. On the fourth day she had a formed stool and passed a very good day. She was up on the eleventh day in a chair and was discharged on the fourteenth day. One year later, well.

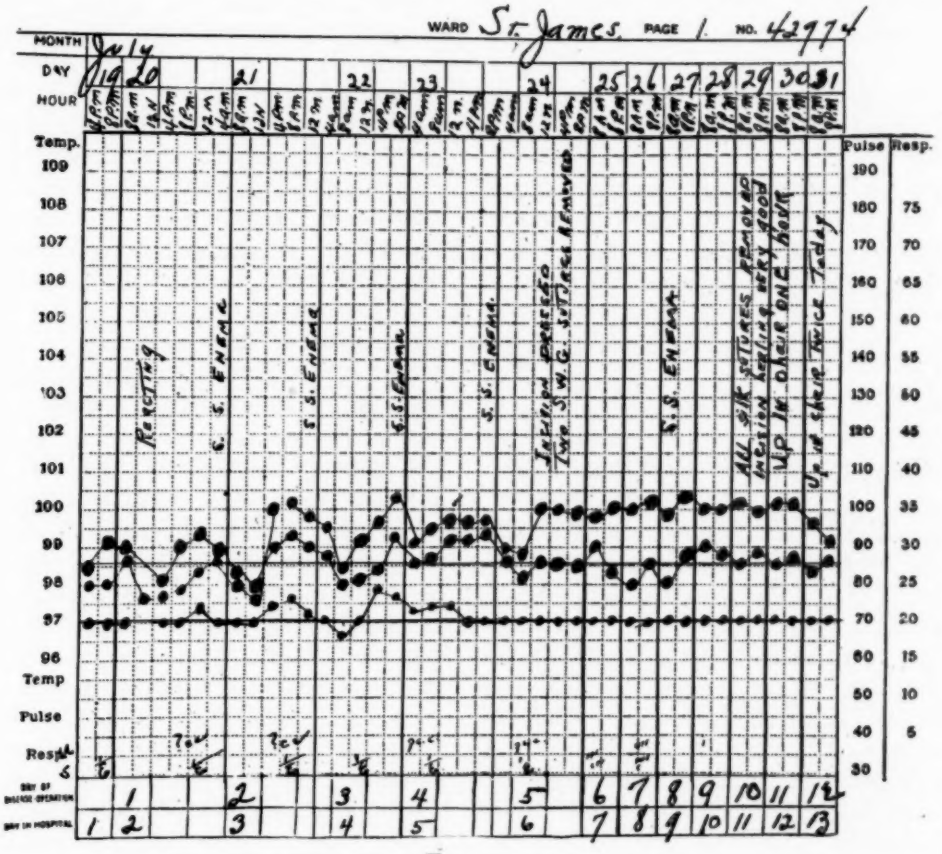

Fig. 1.

Case II. St. Agnes' Hospital No. 44,930. White, male, aged sixty-four.

Present illness: First attack five years ago. He became unconscious while eating and remained so for one hour; was in convulsions. He foamed at the mouth but did not bite his tongue. There were premonitory symptoms; he felt dazed following the attack. Two years ago he had a similar attack and a third one the night before admission. He went into convulsions; there was excessive saliva from the mouth but he did not bite his tongue. This third attack lasted one half hour but he was dazed for an hour after regaining consciousness. He had been having frontal headaches for two weeks and shooting pains in the occipital region. Vision became blurred momentarily.

Past history: Usual childhood diseases. No serious illness. Seen in consultation two years ago for kidney colic. Had been treated for ulcer of the stomach; was undernourished. Seen again in March 1928, complaining of heart attack following acute indigestion. Had an attack of angina pectoris following the first attack of unconsciousness; two days before admission he had a smothering sensation in the upper thorax after exertion.

Family history: Mother died of tuberculosis, aged sixty-three. Father died of apoplexy, aged sixty-one. Three sisters died of tuberculosis. No history of epilepsy in family.

Physical examination: Well developed and nourished white male. Heart slightly enlarged, sounds regular in rate and rhythm but very feeble. No apparent murmurs. Abdomen, moderately obese. On palpation there is slight

tenderness over McBurney's point. Dullness, on percussion, slightly below the costal margin and to the right of the ensiform cartilage. Liver is not enlarged. Moderate arteriosclerosis. Blood pressure; 148/100; blood count, R.B.C. 5,560,000. W.B.C. 10,600. H.G.B. 85; blood sugar, 125 mgs., Wassermann, negative; urea, 36 mgs. normal of 18 mgs; urine, normal. Few epithelial cells.

Roentgen-ray Examination: March 17, 1928. Gall-bladder region. After the oral ingestion of sodium tetraiodophenolphthalein, fourteen and one-half hours after the ingestion of the dye and two hours after eating, the gall bladder was not visualized. There were no positive stone shadows.

Fluoroscopical examination of the chest shows moderate dilation of the arch of the aorta. Heart not enlarged. Moderate fibrosis of the lungs.

Diagnosis: Gall stones and appendicitis.

Operation: March 19, 1928. Cholecystectomy, appendectomy. (Dr. Mortimer. Assistant, Dr. Matthey. Ether, Dr. Keech.)

Operative note: Incision was begun at the costal cartilage, down following the free border of the ribs to the mammary line and then down approximately 3 in. Fascia was separated crosswise; muscles separated by blunt dissection; peritoneum opened with longitudinal incision. There were many adhesions in the region of the gall bladder and it was difficult to locate. On palpation we found what we thought to be the gall bladder imbedded in the liver. Grasping the fundus with an Allis clamp, but still in doubt of the mass, we made an opening and removed one large stone (the size of a guinea hen's egg), a number of small stones and considerable mucus. The serous covering of the gall bladder was stripped down to the cystic duct and a sound passed through the cystic and common ducts. No adhesions or obstruction found. The cystic duct was tied and amputated near the common duct. No. 0 catgut was used. The serous covering of the gall bladder was replaced with three stitches. Large and small packs were removed.

The left hand was then passed to the region of the cul-de-sac and the appendix brought into view. Mesoappendix and all adhesions were tied. A Pagenstecher suture was applied and the appendix amputated with cautery; stump was inverted. Abdomen was closed through and through with No. 2 catgut; silk was used in skin and five silver wire sutures through skin and fat for reinforcement. Patient was given 1000 c.c. of saline while on the table.

Gross pathology: The specimen consisted of a gall stone about the size of a marble, egg-shaped, corrugated, very friable. On section it was crystalline and golden yellow.

Postoperative note: The day following operation there was no nausea and the patient had passed a comfortable night. On the second day he passed flatus. He was very comfortable and desired nourishment. Given soft diet in seven days and after ten days was on regular diet. Up in a chair on the fifteenth day and discharged from the hospital on the twenty-first day in excellent condition.

Three months later. Patient reported he is feeling better than he has felt for ten years.

Case III. St. Agnes' Hospital No. 44,799. White, female, aged twenty-seven, married.

Present illness: Referred for vaginal odor and discharge. Pain under right scapula; tenderness over the gall bladder; vomiting. Pain comes on after eating; stools have been clay and slate colored.

Past history: Laporotomy three years ago. Appendix, both tubes and part of both ovaries removed. The gall bladder was then found full of stones but owing to the patient's condition the stones were not removed. Was advised to have hysterectomy in two years. I did a currettage and cauterization of the cervix and the patient was relieved for four months, but the odor then returned and was even greater. By this time she had attacks of gall-stone colic which were so severe she consented to another operation.

Family history: Mother living, had a hysterectomy at age of forty-seven. Otherwise history is negative. Menses began at age of thirteen. Missed half of the time; irregular; painful. Relieved by operation for about one year but then trouble returned. Marital: One child six years old. She had one miscarriage about six weeks before admission.

Physical examination: There is tenderness over the entire abdomen which is more marked over the region of the gall bladder. No rigidity; no palpable masses; no jaundice. Blood pressure, 132/75; blood count, R.B.C. 4,120,000; W.B.C. 10,400; H.G.B. 85.

Diagnosis: Gall stones. Degenerated uterus.

Operation: February 27, 1928. Cholecystectomy. Hysterectomy. (Dr. Mortimer. Assistant Dr. Matthey Ether, Dr. Keech.)

Operative note: The incision was begun at the ensiform cartilage, carried down the lower border of the ribs to the mammary line and then down one-half inch. The gall bladder was easily located. The fundus was grasped with an Allis clamp and the surrounding viscera packed off. The serous covering of the gall bladder was incised, encircling the fundus, stripped back to the cystic duct and then the duct was clamped. A small incision was made into the cystic duct and a probe passed down into the common duct. No obstruction found. The cystic duct was tied and anchored with No. 2 catgut and the gall bladder amputated intact. The stump was reinforced with two catgut sutures. The serous covering was drawn over the stump and secured with three catgut sutures. The wound was closed in the usual manner with No. 2 catgut for deeper sutures, silk in skin and reinforced with two silkworm gut sutures.

Mid line incision was then made parallel to the old scar and a hysterectomy done in the usual manner. The patient stood the operation very well. Was given 1000 c.c. of normal saline subcutaneously while on the table.

Post operative note: After twenty-four hours the patient had had a fairly good day; some pain; fed by Murphy drip; voided every four hours; given small doses of morphine. On the second day, expelling flatus. There was slight headache and some nausea. Given ½ grain of codeine. Light diet on the third day. In six days the patient was comfortable on light doses of codeine. Out of bed on the twelfth day for an hour. Discharged from the hospital on the sixteenth in good condition.

Case iv. (Case 1. NGBS. March, 1915.) White, female, aged twenty-six, married. Two children.

Diagnosis: Acute cholecystitis. Cholelithiasis.

Operation: March 15, 1925. Cholecystectomy. (Dr. Mortimer.)

Modified Mayo incision. The gall bladder had ruptured but was well walled off. The remnant of the fundus was grasped with an Allis clamp and the surrounding area walled off with gauze packs. Fifty-two stones were removed. The serous covering was stripped back to the cystic duct. The cystic duct was ligated and anchored; the serous covering was then drawn back. A rubber drain was placed in this field and the abdomen closed in the usual manner with No. 2 catgut for deeper layers and silk in skin.

Post operative note: The drain was removed on the fifth day. On the twelfth the patient was discharged from the hospital in good condition, walking.

Thirteen years later, the patient reported that she has been well and has had three children since the operation.

SUMMARY

Subserous enucleation of the gall bladder by Doyen's method is advantageous for the following reasons:

1. It gives an excellent exposure to the cystic duct, rendering confusion with the common duct impossible.
2. It requires no traction on retractors.
3. It leaves no raw surfaces for adhesions to form.
4. It permits removal of the gall bladder and contents intact.
5. It permits probing of the common duct for stones and stricture.
6. It does not necessitate tying the cystic artery.
7. It causes no injury to the bed of the liver and there is no hemorrhage from this point.
8. It requires no drainage.
9. The lack of trauma insures a comfortable and speedy recovery with the minimum amount of shock to the patient. Absence of nausea and pain.
10. The modification of Mayo's incision in conjunction with the pad permits a better exposure without retractors which causes less need for packing and in turn less trauma to the celiac plexus.

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CASE REPORTS

SUPERNUMERARY GALL BLADDER*

WILLIAM L. WOLFSON, M.D., F.A.C.S.

BROOKLYN

THERE are many anomalies of the gall bladder and ducts. Such structural deviations, while interesting anatomically, also may have considerable clinical and surgical importance.

Cases of accessory gall bladder are extremely rare. In an exhaustive study of the clinical, autopsy and comparative anatomical data on the subject, Boyden in 1926 found that while the condition is relatively common in certain animals, notably cats, only 20 instances in human beings had been reported between 1674 and 1926.

In considering these 20 cases of accessory gall bladder, Boyden classified them as follows: (1) vesica divisa, or bilateral gall bladder; 2. vesica duplex, or double gall bladder, with two cystic ducts. The latter was further subdivided as: a, Y-shaped, in which the two cystic ducts unite before entering the choledochus, and the gall bladders, usually adherent, occupy the same fossa; b, ductular type, in which the two cystic ducts empty separately into the common bile duct. The two gall bladders, generally separate, may occupy different lobes.

Of the ductular type there are only 4 represented in the series. The earliest account was by Blasius in 1674. A possible instance was reported in the *Philosophical Transactions*, 1693–94. The next example was observed by Purser in 1886. The fourth case was reported by Professor Lambert of the University of Alabama in a personal communication to Dr. Boyden. He identified two separate bladders, the accessory one being an "outgrowth of the common duct in the form of a small saccular appendage presenting all the structural conditions of the gall-bladder." It was located half a centimeter or more from the junction of the main cystic duct with the choledochus.

Sherren, in 1911, Schachner in 1916 and Nichols in 1926, described double gall-bladders, some containing stones; but the exact terminations of the two cystic ducts were not revealed.

The following case is reported as a supernumerary gall bladder of the ductular type, making the fifth of that classification.

CASE REPORT

History: The patient, female, aged nineteen, was admitted to the Jewish Hospital of Brooklyn, May 2, 1927, complaining of intermittent pain in the right hypochondrium over a period of nine years.

At the age of ten the patient first experienced slight gnawing pains in the upper part of the "stomach." These early attacks lasted but a few minutes. During that first year she had four distinct attacks. They recurred through the next two years with increasing frequency and severity. Several radiographic studies were made of the gastrointestinal tract and of the gall bladder. No abnormality was demonstrated.

Four years later, at the age of fourteen, the patient had an attack of epigastric pain that persisted for an hour. It was diagnosed as an inflammation of the gall bladder. The following year and a half the attacks continued with less severity, but with distress after eating and a sense of fullness as from undigested food. A roentgenological examination then made disclosed possible gall-bladder calculi, and an operation was advised.

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At sixteen the patient had the most severe attack of pain which lasted seven hours and was associated with vomiting of a greenish

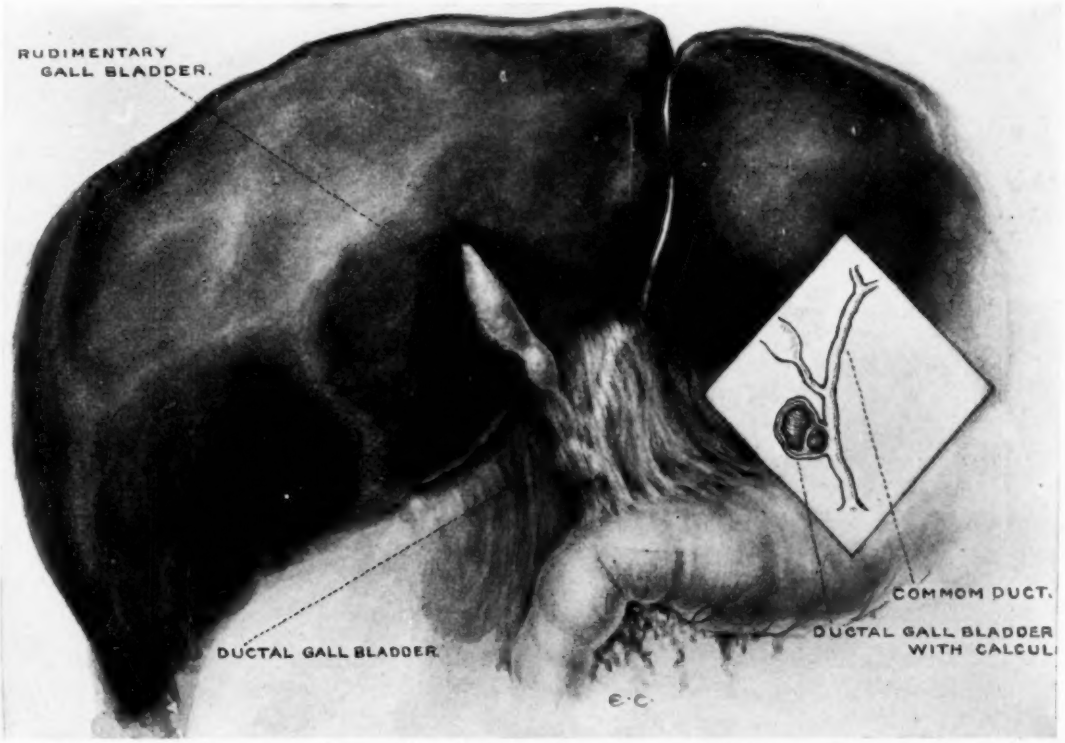

Fig. 1. Showing the position and relative size of the rudimentary gall bladder to the supernumerary (ductal) gall bladder.

fluid. This experience was repeated the following night for two hours. Roentgenological investigation of the gall bladder failed to show any calculi. These attacks of pain accompanied with vomiting recurred at irregular intervals in undiminished severity.

After every meal for the past year she has complained of belching, sour eructations, epigastric distress, fullness, pyrosis and bloating. The roentgenographical studies now made of the gall bladder and ducts by the dye method revealed 2 calculi.

Physical examination: The physical examination showed a well-developed, well-nourished female. The only significant finding was a slight tenderness in the right upper quadrant.

Operative Findings. Through an upper right rectus incision the following findings were noted: A rudimentary, narrow gall-bladder, 4.8 cm. in length, lay deeply set in the gall-bladder fossa (Fig. 1). It had slightly thickened walls and contained a dark viscid bile, but no calculi.

The cystic duct, 2.4 cm. in length and 16 cm. in diameter, opened into the common bile duct at a slight angle. About 1.2 cm. below its entrance was a sac, approximately 1.5 cm. in

Fig. 2. Microscopic section of supernumerary gall bladder. The inner surface, the mucosa, is lined by columnar epithelium and assumes a papillary formation. Many of the tubular glands extend into the muscularis and serosa.

length and .8 cm. in diameter, arising from the common duct and lying close to the posterior

parietes, but distinctly free from the liver. The wall was surrounded by perisacular adhesions, from which it had to be dissected. Contained therein were 2 distinct rough calculi, yellowish brown in color, one of which was flush with the common bile duct (Fig. 1).

Cholecystectomy was performed with ligation of the cystic duct near the common bile duct. After incising the ductal sac and removing the calculi, the sac was cut away close to the common duct and drainage was established with a 16 F. catheter through the opening made by the removal of the sacular appendage.

Pathological Report. The specimen consisted of a sac 0.8 × 1.5 cm., its surface pink in color, smooth and glistening, with a small node 1 cm. from one end. On section the wall was found to be thick, the colorless mucoid secretion containing a small calculous deposit about 0.2 cm. in diameter.

The microscopical examination showed the specimen to consist of an outer thick serous membrane, covering a layer of smooth muscle arranged in irregular bundles. On the inner surface was a mucosa made up of papillae, lined throughout by columnar epithelium (Fig. 2). Many of the tubular glands extended into the muscularis and serosa. The small node was the seat of a diffuse endometrial hyperplasia of an inflammatory nature.

The patient made an uneventful recovery and was discharged sixteen days after operation. When recently seen, she had improved considerably in weight and was free from any digestive or upper abdominal distress.

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CASE REPORTS BY DR. STANLEY R. WOODRUFF*

NEW YORK

MULTIPLE CALCULI IN LEFT PELVIS OF HORSESHOE KIDNEY

THE patient, a female, aged thirty years, married, white, had suffered for the past five years with intermittent attacks of pain in the left flank, sometimes accompanied by chills and temperature. She had had long continued attacks of indigestion and malaise, and had reached the state of discouragement at which most chronic invalids usually arrive. The renal history had been varied. She had at times complained of frequency and some dysuria, and as a usual thing there had been little attention paid to the urine. She had had three abdominal sections for various preoperative diagnoses. Her appearance was that of a rather delicate, pale, undernourished woman. There was a slight systolic heart murmur. The lungs were normal; the abdomen was flaccid and revealed a mass in both the right and left umbilical regions, the one on the left side being larger. It was only slightly movable, and there was no complaint of tenderness. The urine had a specific gravity of 1012, was cloudy in appearance, and contained many pus cells and considerable albumin. The preliminary roentgenogram revealed several fairly distinct shadows grouped much lower and toward the midline than would be usually considered as being renal calculi. The kidneys were not definitely shown. By cystoscope the bladder was seen to be entirely normal. A renal functional test with indigo carmine showed an appearance time of ten minutes from the right ureteral orifice and fourteen minutes from the left, the quantitative output being practically equal from both sides and about 50 per cent of normal. Catheterized specimens from both sides showed a preponderance of pus from the left kidney. There was, however, considerable pyuria from the right side. Ureteropyelograms were made at separate sittings of both sides and the typical appearance of horseshoe kidney was demonstrated. The shadows observed in the preliminary exposure

* Read before Section of Genito-Urinary Surgery, New York Academy of Medicine, April 18, 1928.

were noted to be covered by the contrast substance in the renal pelvis of the left kidney.

At operation the ureter on the left side was found to enter the renal pelvis at its upper third, and most of the calculus deposit had settled into its extreme lower end which had become practically a cul-de-sac. A heminephrectomy was hardly indicated here on account of the poor function of the right portion, and a plastic reimplantation of the ureter into the lower portion of the left renal pelvis was successfully accomplished.

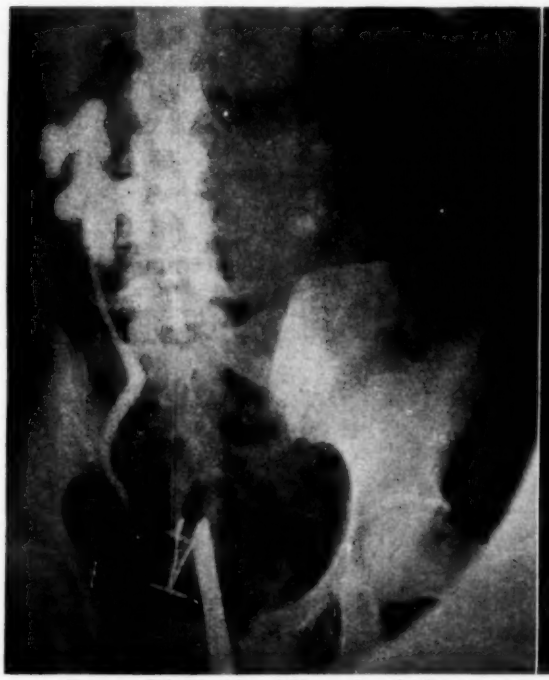

Fig. 1. Right side of horseshoe kidney.

Fig. 2. Left side of horseshoe kidney.

RENAL, URETERAL, VESICAL AND IMPACTED URETHRAL CALCULI SIMULTANEOUSLY IN SAME PATIENT

THIS patient, a male, aged thirty-four years, married, white, entered the hospital acutely ill, complaining of severe pain in the penis, and great difficulty and pain in attempting urination. He gave a history of passing renal calculi on several occasions during the last three years. His attack of pain at this time came on three days ago, during the act of micturition, and was accompanied by sudden stoppage of the flow. Urination has been increasingly painful and difficult up to admission, when it became necessary to anesthetize the patient in order even to make an examination. At this time a hard, calcareous object was made out both by palpation and instrumentation to be impacted in the urethra at about the penoscrotal junction. After considerable difficulty, this was removed and a catheter passed

Fig. 1. Pyelogram of case of renal, ureteral, vesical and impacted urethral calculi.

into the bladder for drainage. The patient was then placed in bed and five days later roentgenogram was taken in order possibly to demonstrate more calculi. The resultant exposure justified the suspicion in that multiple calculi were demonstrated in the left kidney, the left lower ureter and bladder. A cystoscopic examination was then made which showed the bladder slightly congested, with tumefaction and edema at the left ureteral orifice. There were three small calculi in the vesical cavity. A renal functional test gave a normal appearance time and normal quantitative output from the right ureteral orifice. There was no appearance from the left side after fifteen minutes' observation, and a small catheter passed to the left renal pelvis revealed no dye, the resultant secretion being a purulent fluid.

The necessity of nephrectomy, ureterotomy, and cystotomy was apparent, and was successfully performed with the removal of all calculi three days later.

CASE REPORTS BY DR. A. R. STEVENS*

NEW YORK

TRANSPLANTATION OF URETERS INTO THE BOWEL

IT has seemed to me worth while to show two cases of double implantation of ureters into the bowel, done by different methods, both because of incontinence but due to different underlying causes.

Case 1. A colored man, aged fifty years, gave a history of having had gonorrhoea several times. In 1914 he had a perineal incision for perineal abscess and stricture, and in 1923 an external urethrotomy and resection of perineal fistula. In July, 1926, he was admitted to Bellevue Hospital because of recurrent fistulae and partial incontinence of urine; in fact he was completely incontinent while standing but only partially while lying down. The Wassermann reaction was negative. His spinal fluid reaction was negative. Sounds up to 30°F. were easily passed to the bladder.

He was operated on in the hope first of cleaning up his perineum. Some of his fistulae were excised and the others opened up into one common perineal cavity. Incidentally, an abscess of the prostate was opened. His wound remained infected and on August 13, a suprapubic cystoscopy was done, in the hope of hastening the healing of the perineal wound by keeping it dry.

The writer first saw the case September 15, 1926. He had then a large perineal wound which was continuously wet, his urethra was open for 3 in., and 1½ in. of the urethra had entirely sloughed. An attempt at cystoscopy was made but the bladder would not hold any fluid. The patient was kept around the ward for a long time in the hope that by care and frequent cleansing we could heal the perineal wound and later make a plastic perineal repair of the sphincter and urethra. However, he did not seem to make any great headway, and the writer suggested to him that, inasmuch as he could not work and associate with his friends in his present condition, he have his ureters implanted in the lower bowel.

On December 17 we operated through a low left rectus incision, located his left ureter, exposed that from the promontory downward, freed the peritoneum, stitched it up behind the ureter, and implanted the latter in the sigmoid by the Coffey technique. No catheter was left in the ureter, but a tube was kept in the rectum for about four days. We were very much surprised that there was no kidney pain following this and no temperature above 100°F. Ten days afterward we injected indigo carmine intravenously and obtained a very marked color in the urine from the rectum.

On January 18, 1927, under spinal and general anesthesia, a mid-line incision was made, the right ureter was exposed from the promontory to the bladder. The bladder was incised and was found to be a real contracted bladder with a wall 1 cm. thick. A ring of bladder mucosa was cut away about the ureteral orifice

* Read before Section of Genito-Urinary Surgery, New York Academy of Medicine, May 16, 1928.

the bladder was sewed up and the right ureter implanted by the Coffey technique. A catheter was left in the ureter. In comparison with the operation on the other side, with no catheter in the ureter, there was some fever (up to 102°F.) and pain in the corresponding kidney. In both instances a cigarette drain was placed at the site of the ureterosigmoidal anastomosis.

About six weeks after this last operation the patient was able to hold the rectal contents for about four hours. When he went home on March 16, about two months after his last implantation, he was able to hold his urine from four to six hours. We did a phthalein test and obtained from the rectum 20 per cent in two hours. His blood chemistry was normal. His perineal wound has not yet closed up.

Case II. The other patient was a case of ectopia vescicae. When the patient first came to the writer in the fall of 1911, he was a boy of sixteen. Three attempts had been made to close the bladder when he was about two years old. It is not necessary to go into the details of the history; they have already been published. We catheterized the ureter and proved that there was no infection of either kidney; the phthalein output was 30 per cent in one hour. Operation was performed April 6, 1911, by the Bergenhem method, that is, both ureters were isolated extraperitoneally from the promontory of the sacrum to the bladder and cut away with a small ring of bladder mucosa attached to each, without either ureteral orifice being cut into. These were implanted into his rectal wall through independent small holes and were left there without any suture. During convalescence there were attacks of pain in the left kidney, which clearly indicated some infection of that side. The patient went home from the hospital two months after his Bergenhem operation, able to hold his urine in the rectum for from two to six hours. For the first few months he had quite a bit of nocturnal incontinence but gradually gained complete control. He called on me last fall and at that time was quite able to hold it for from four to six hours and had measured individual voidings that were as much as 350 c.c. His phthalein output in two hours was 25 per cent. He has gone on now about seventeen years and is entirely comfortable and able to work and mingle with his friends.

LEUCOPLAKIA IN A BLADDER DIVERTICULUM

THERE are four interesting points in this case: (1) large chunks of epithelium were passed; (2) the patient had no urinary complaint; (3) he had leucoplakia in this diverticulum, and (4) there was a cystoscopic resemblance to carcinoma.

There had been only two cases of bladder diverticulum containing leucoplakia reported up to the time when Dr. Bughee reported one and the writer reported this at the recent meeting of the American Association of Genito-Urinary Surgeons. The first operation the writer performed on returning from this meeting was another diverticulum case which presented typical leucoplakia both grossly and microscopically.

The patient had a very marked urinary infection and complained of marked frequency of urination.

DERMOID CYST OF KIDNEY*

J. J. Valentine, M.D., F.A.C.S.

NEW YORK

ONLY a few cases of dermoid cyst of the kidney have been reported in the literature and their pathological study is scantily recorded. The most recent contribution seems to be by J. F. Baldwin of Columbus, Ohio in 1915. In his article he quotes the famous case of Sir James Paget[1] of dermoid tumor of the kidney in

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the sheep, as well as five complete case reports in the human.

In order to analyze these cases and to compare the interesting findings with the one now presented, they may best be tabulated.

DERMOID CYSTS OF KIDNEY

Cases	Age	Sex	Palpable Tumor	Urinary Symptoms	Pain	Preoperative Diagnosis	Pathology and Contents of Cyst	Result
Haeckel's[3] reported by Wedeman.	58	F	Several yrs.	None	Yes	None made	Pultaceous yellow substance. Hair	Recovery
Goldsmith[4]	20	M	2 yrs.	Hematuria	Colic	Floating kidney with twisted pedicle	Subcapsular cyst. Large quantity reddish mealy material. Hair	Recovery
Walker[5]	11	F	1 yr.	Irritable bladder	Colic and pain in l. lumbar region	None made	Three cysts; one filled with fatty material and fine hair	Recovery
Schlegtendal & Madelung.[6]	22	M	All his life	None	None	Ecchinococcus cyst of liver	Cystic tumor filled with smeary mass. Fat cells. Crystals of cholesterin	Died
Boni[7]	45	F	10 yrs.	Not stated	Not stated	Hydronephrosis, probably due to calculus	Five intercommunicating cysts. Yellowish green turbid fluid and detritus. Skin structure with sebaceous sweat glands. Hair follicles and hair	Died
Wyss[8]	..	..	.....	.....	.....	.....	Ossified walls containing mass of cholesterin	
Bardenheuer[9]	..	..	.....	.....	.....	.....	Fat and hair	
Baldwin[10]	16	F	14 yrs.	None	None	Possible ovarian dermoid	Walls of bony plates. Cavities containing different colored fluids. Cholesterin crystals. No hair	Recovery
Author's Case	46	F	No	Yes	Yes	Probable renal calculus (cortical)	Subcapsular cyst. Thick light yellow pasty substance. Fat globules and cholesterin crystals. No hair	Recovery

It will be noted that a palpable tumor occurred in all but the present case, and that the tumor had been there for several years.

Probably because these cases presented themselves many years ago and because they were studied by other than urologists, no detailed urological investigation was made either by cystoscopy or roentgenology.

The pathological findings, while not alike in all the cases, bear some degree of uniformity. For example, hair was found in the cysts five times. An indefinite substance of varying color and consistency occurred within the cysts in 6 cases. Cholesterin crystals were found in 4 cases; fat in 4 cases. Two of the cysts were subcapsular.

Attention is called to the fact that in no instance was it possible to make a correct preoperative diagnosis, as there seemed to be no definite characteristic clinical symptoms nor has there been a sufficient number of cases studied from the urological standpoint to submit additional valuable data.

CASE REPORT

Mrs. M. O., aged forty-six, admitted to Polyclinic Hospital June 23, 1927. Chief complaints were frequency and burning urination, pus in urine for many years and pain in back.

Her general history was negative except for her urological disturbances. For many years she had had marked frequency of urination with considerable burning which occurred during and after the act. At times she had seen a little blood in her urine. She also stated that she had had a left-sided backache for several years.

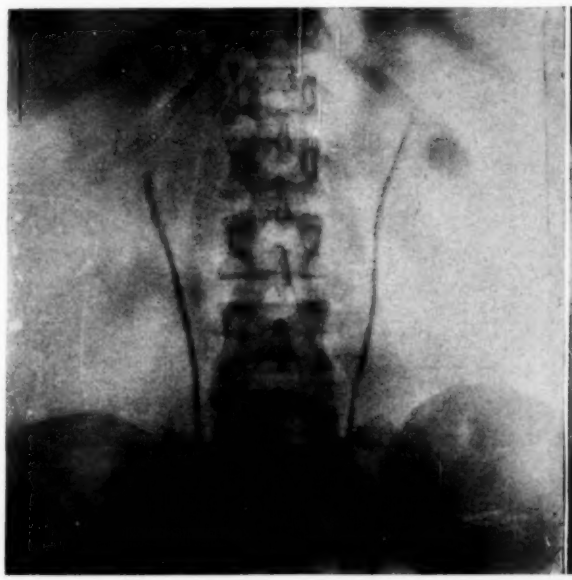

Fig. 1. Showing shadow in left kidney resembling calculus.

Fig. 2. Pyelogram showing shadow within kidney.

General examination negative as regards chest and abdomen. Because of her marked frequency and burning urination, our attention was first fixed on her bladder and urethra. Voided urine was hazy, containing a moderate amount of pus, a few red blood corpuscles, and culture showed a gram-negative bacillus growth.

Cystoscopy showed bladder congested, otherwise negative. Not finding sufficient pathology in the bladder, urethroscopy was performed and a small cystic tumor was seen in the midline on the upper margin of the bladder neck. Also several small cystic bodies were seen to project into the urethra anterior to the sphincter. These bodies as well as the larger tumor were destroyed by fulguration with the electrotome current. Following this there was very little reaction. The patient remained in the hospital about forty-eight hours and was allowed to go home.

She reported to the Clinic in two weeks and stated that her frequency of urination was materially reduced and that she felt very much better. She had less burning than she had experienced in years. Her backache, however, was not in any way relieved. A plain roentgenogram was taken and cystoscopy performed at this time to investigate her upper urinary tract.

Both ureters were catheterized and specimens obtained. Examination of these specimens showed:

	Left	Right
Gross appearance of urine	Clear (later bloody probably trauma)	Clear
Urea	0.4 per cent	0.9 per cent
Blood	5–10 per field	10–15 per field
Epithelium	Rare cell	Occasional
Leucocytes	About 2 per field	1–2 per field
Culture	Sterile	Sterile
Indigo-carmine	4 min. in good concentration	4 min. in good concentration

Blood chemistry, normal.

Despite the lack of pathology in urine, the roentgenographic findings, with and without pyelograms, made us conclude that we were

dealing with a cortical stone in her left kidney, and exploration was advised.

Operation. A left curved, oblique, lumbar incision was made, exposing a normal sized kidney. Before freeing kidney to bring it up into the wound, careful palpation was made for the calculus but it was not felt. The kidney was then freed and brought up into full view in the wound. Inspection showed the true capsule of the kidney to be separated from the kidney itself by fluid under the capsule. Several bubbles could be seen under the capsule and made to move by making pressure on the kidney. At about the juncture of the upper and middle portions of the kidney, in the central line along the cortex, could be seen a yellowish mass, plainly visible through the true capsule and not attached to it. The true capsule was nicked and about 2 drams of fluid escaped. The true capsule was separated for the entire length of the kidney and was readily peeled away, exposing the growth just mentioned. This growth, as one looked down on it, appeared larger than the diameter of a twenty-five cent piece, and looked something like an old tuberculous (firm, cheesy) deposit. It seemed to be covered by its own capsule, and it was easily separated from the adjacent cortex of the kidney. It was not attached to the true cortex in any portion except at its very base where it seemed adherent. When enucleated from its bed the point of attachment bled freely. This point of bleeding was clamped and ligated. The bed of this growth now seemingly was a gap in the kidney, lined with true kidney cortex. The capsule was then sutured with plain catgut and the cavity from which the growth was removed was packed with iodoform gauze, the end of which was used as a drain extending from the upper angle of the wound, and the kidney was dropped back into its natural bed. The wound was closed in the usual layer fashion.

FIG. 3. Schematic drawing showing subcapsular location of cyst and its relation to calcies.

FIG. 4. Dermoid cyst after its enucleation from kidney.

Postoperative recovery was uneventful and the patient left the hospital twenty-six days later.

Pathological Report. The specimen was composed of a cystic structure removed from the kidney. It was circumscribed, ovoid in shape, and measured $3\frac{1}{2} \times 2\frac{1}{2} \times 1$ cm. It contained a thick, light yellow, pasty material.

Microscopical examination of this material showed numerous fat globules and a few cholesterin crystals. The wall varied from 1 to 3 mm. in thickness and the inner surface was roughened. There was apparently no lining.

Microscopical examination of sections taken from different areas of this wall showed that it was composed chiefly of dense strands of hyaline connective tissue. On the inner surface there was a loose areolar structure in which there were many blood vessels, some of which had rather thin walls but others had very much thickened walls. It was also infiltrated with numerous inflammatory cells. There was no lining epithelium observed. Diagnosis was dermoid cyst of the kidney.

Postoperative history. Patient has been seen frequently since the operation, and she states that she has completely recovered from her urinary frequency as well as from her backache.

The case in question is interesting primarily because of the early and accidental

discovery of the cyst in the course of ordinary routine urological examination. The cyst, casting a roentgenographic shadow which was interpreted as being a calculus, was sufficient reason for operative interference for the relief of pain. It is likewise of interest to have learned that the cyst was subcapsular, causing an exudate to distend the true capsule of the kidney, producing nephralgia. After removal of the cyst the pain ceased.

J. R. Losee, who studied this specimen, refers us to Ewing's[10] work wherein is recorded: "Epidermal rests derived from the Wolffian duct are probably the source of certain dermoids of the kidney. They contain no dermal glands."

Rokitansky[11] says there is often no epithelium in the larger cysts and their inner layer is striate blastema externally spreading into fibers in the direction of the long axis of the oval nuclei it contains.

Losee states that regardless of the fact that no squamous epithelial lining was observed in this cyst, he believes the diagnosis can be made from its contents.

It is understood that these cysts arise from remnants of the mesonephros, which structure is supposed to degenerate in the latter stage of the development of the embryo. If part of this does not degenerate there is some form of cystic structure remaining. The epithelia, lining these mesonephric tubules, are not very well differentiated at that early period, therefore one can conceive that it would be possible to have any one of these various types of epithelia.

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CASE REPORTS BY DR. ARTHUR M. DICKINSON*

ALBANY, N. Y.

BILATERAL SNAPPING HIP

SNAPPING of the hip may not be an unusual condition in large orthopedic centers, but to the general surgeon it is a very uncommon and often trying condition. References to it in the literature of this country are rather meager as contrasted with the quite frequent cases reported in foreign literature.

A certain amount of snapping of the hip may be almost physiological in children and also in adults with relaxed periarticular structures. In some instances it becomes a practiced trick to be demonstrated at will for the astonishment of companions. As a rule this type of snapping hip does not cause pain and discomfort. When pain and discomfort do result, the condition becomes pathological. Commonly only one hip is affected.

ETIOLOGY

There are many factors which have been considered as causes in producing snapping of the hip. A few of these are inflamed bursae, malformations of the hip joint, a tic or spasm of the muscles, either voluntary or involuntary, and muscular relaxation, especially of the gluteus maximus, with or without actual tears in the muscle.

Before the causal factors are weighed, it is well to consider the anatomy of the parts. The hip joint is essentially a ball and socket

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joint, the head of the femur being received in the acetabulum. Both head and acetabulum are covered with cartilage. The cartilage of the acetabulum is horseshoe shaped, being deficient below. The ligaments of the joint are the capsular, the iliofemoral, the teres, cotyloid and transverse. Many bursae of varying sizes exist about the joint, some anatomists having described twenty-one. The bursae that are of practical interest to us at this time are those located in the neighborhood of the greater tuberosity. There are several of these. One is located between the greater tuberosity and the gluteus maximus, another between the tuberosity and the skin, two between the tuberosity and the gluteus medius, and another between the tuberosity and the gluteus minimus. The muscles and fascia most concerned in snapping hip are the glutei maximus and medius and the iliotibial band. The gluteus maximus is the very fleshy, strong muscle which forms the prominence of the buttocks. It arises from the ilium and sacrum and, passing obliquely down and outward, is inserted by a thick tendon which runs across the greater trochanter into the fascia lata covering the outer side of the thigh. Some of the deeper fibers are inserted below the greater trochanter. The gluteus medius arises from the ilium and is inserted into the oblique line which traverses the outer surface of the greater trochanter. The iliotibial band is that thickened portion of the blended layers of the fascia lata which continues down the thigh to be inserted into the external tuberosity of the tibia. The greater trochanter is a large irregular bony eminence situated at the outer side of the neck of the femur. It is directed outward and backward. The gluteus medius tendon is attached to its external surface and the tendon of the gluteus maximus plays below and behind.

It will be seen from this brief review of of the anatomy that snapping of the hip must arise either in the hip joint or in connection with the bursae and muscles and the greater tuberosity. In all reported cases of true snapping hip in which roentgenographic studies of the hip joint have been made, the bony parts have been found normal. It is difficult for one with any semblance of mechanical understanding to conceive how an enlarged or inflamed bursa could be the cause of snapping of the hip. In examining the patient with a snapping hip, one is at once struck with the impression that the snap occurs in the neighborhood of the greater trochanter. It seems as if there was a tight band which snapped across the trochanter when the thigh was rotated. The two structures which could possibly cause this are the gluteus maximus and the iliotibial band. The majority of writers consider the latter as the chief offender.

Binne[1] in 1913 reported 2 cases, in 1 of which there was found a sausage-shaped thickening of the fascia lata and in the other the snapping part was the anterior border of the gluteus maximus. Mayer[2] in 1919 reported 4 cases. In case I he stated there was a snap audible to the distance of 20 to 30 feet. It was successfully treated by adhesive plaster strapping with a pad behind the trochanter. Cases II and III were treated by a special pelvic girdle. Case III was due to previous removal of a strip of fascia lata by himself. Case IV was operated upon; no thickening was found in muscle or fascia. The edges of the fascia were sutured to the periosteum of the greater trochanter and a cast applied. Mayer places great stress upon the thickening of the portion of the fascia lata which Zur Verth called the tractus cristofemoralis. Mayer feels that the snap is the result of this band catching behind the trochanter and that the band jumps when the strain becomes too great. He is apparently of the opinion that the condition is solely the result of fascial changes. He concludes that a relaxation of the gluteus maximus is a necessary factor in the causal mechanism.

Jones[3] in 1920 cited 2 cases in both of which the snapping was due to the tendon

of the gluteus maximus. He found that the snapping occurred only when the tendon was tense. He noted that the snap disappeared when the patient was relaxed by an anesthetic. Pruitt[4] in 1920, reporting on this subject, stated that all cases were due to some abnormality of the fascia lata or the greater trochanter whereby the fascia was caught behind the trochanter. He considered as the etiological factors the thickening of the tractus cristofemoralis and unusual prominence of the greater trochanter. Fairbank[5] in 1921 reported briefly a case of bilateral snapping hip complicated with other orthopedic conditions. Lovett[6] states that the condition is one of no great rarity and that the common cause is a slipping of a tendinous band (usually the iliotibial) over the greater trochanter. DaCosta[7] says that it is due to a slipping of a fibrous band (iliotibial) over the greater trochanter. Whitman[8] considers that the snap is due to friction between the tendon of the gluteus maximus and the greater trochanter. The weight of evidence seems to favor the iliotibial band and the gluteus maximus as the causative factors. Whether the condition is congenital or whether it is developmental or traumatic cannot be stated with assurance. However, it could be explained on the basis of a congenital defect more easily than by other means.

SYMPTOMS AND PHYSICAL FINDINGS

Patients with this condition commonly report that they have a snapping hip. The diagnosis simply requires confirmation. While snapping hip may not be a disabling condition it is uncomfortable for the patient. Some patients state that the snapping causes a sick feeling in the stomach; others tell you that there is simply a feeling of weakness in the affected hip. With a few individuals, particularly children, it is simply a trick performed at will and causes no symptoms.

The snap can always be felt by the observer; frequently a bulging of tissue may be seen at the time of the snap and in rare instances the snap is definitely audible. Upon palpation, one can feel the band snap as the leg is moved. A very satisfactory manner of feeling the snap is to walk behind the patient as he walks along, keeping a hand over each hip. If the patient reclines on the examining table, external rotation of the thigh with partial flexion of the leg will usually bring out the snap. By placing one hand on the hip and rotating the thigh with the other hand placed on the knee, the snap can be demonstrated. In some instances the snap is so severe that a roll of tissue may actually be seen jumping over the trochanter as the snap occurs. Mayer[2] cites a case in which the snap was audible to a distance of 20 to 30 feet. Frequently we are just able to hear the snap in a quiet room when in close proximity to the patient.

In every case of suspected snapping hip, roentgenograms of the pelvis and hips should be made to rule out changes in the bony skeleton.

TREATMENT

Non-operative treatment has been successful in some of the reported cases of snapping hip. Mayer[2] reported one case treated satisfactorily with adhesive plaster straps and pads placed behind the trochanters. He also reported the successful use of a pelvic girdle in these cases. In the majority of instances however, non-operative treatment has not been a success.

Obviously the object of operation in these cases is to relieve the cause of the snapping. To do this, it is necessary to actually see the band, which is at fault, snap over the trochanter. As the snap is frequently absent under the relaxation due to general anesthesia, operation should be performed under local anesthesia. Various types of operative procedures have been used, depending on what is found at operation. Binne[1] treated his case with a sausage-shaped thickening of the fascia lata by raising the periosteum below the greater trochanter and suturing the posterior edge of the incised fascia lata to this.

The anterior edge was then sutured over. Mayer[2] treated one case by suture of the cut edges of the fascia lata to the greater trochanter. Lovett[6] mentions several procedures which have given relief: suture of the obstructing band to the tendinous insertion of the vastus externus and the neighboring periosteum, uniting the iliotibial band to the femur behind the trochanter, or sewing the posterior edge of the band to the gluteus maximus. Pruitt[4] recommends simple division of the snapping band.

Judging from reported cases, the more complicated procedures mentioned are no more successful than simple division of the band. Furthermore, immobilization of the limb should not be resorted to after operation as was formerly the custom. Walking should be encouraged within a few days after operation. Briefly, then, the successful treatment of snapping hip requires but simple division of the obstructing band under local anesthesia.

CASE REPORT

W. P., male, aged nineteen, entered Memorial Hospital on January 11, 1927, complaining of snapping of the right hip. He had been injured three months previously when he fell several feet, landing on his back. The fall did not incapacitate him, but about three weeks later his right hip commenced to snap. For this condition he had been operated upon on November 26, 1926, but the result was unsatisfactory.

When first seen by the author there was definite snapping of the right hip. The scar of previous operation was well healed. A mass of tissue could be seen to jump forward over the greater trochanter as the patient walked. It could be palpated very plainly but there was no audible snap. With the patient reclining on the examining table, the snap could be produced by partial flexion and external rotation of the thigh. The constant repetition of the snapping made the patient feel sick at the stomach. Roentgenograms of the pelvis and hips were negative for any bony changes. The general physical examination and laboratory findings were negative.

On January 12, 1927, operation was performed under ether anesthesia. Due to the formation of scar tissue from the previous operation, anatomical details were somewhat distorted. An incision 5 in. long and parallel to the long axis of the thigh was made with its center over the greater trochanter. The iliotibial band seemed to be abnormally thickened and tense, so this was divided transversely. The cut edges of the fascia lata were then sutured to the greater trochanter. The wound was closed and a plaster spica applied. The cast was left on for three weeks and when it was removed, no snap could be produced in the right hip. A few days later the patient began to complain of snapping of the left hip. On February 16th the left hip was operated upon under ether and a condition similar to that on the right side was found with the exception that there was no scar from a previous operation. The treatment was the same: section of the iliotibial band with suture of the fascia lata to the greater tuberosity and application of a plaster cast. The cast was removed after three weeks and no snap could be elicited in this hip. The patient was discharged on March 17, at which time the right hip was noted to snap occasionally but not seriously.

On April 6 the patient was readmitted because of a return of the snapping of the right hip. The intensity and frequency of the snap had been increasing since discharge from the hospital until it was now as great as before operation. On April 7 the right side was again operated upon, this being the third operation on that side. This time operation was performed under local anesthesia (infiltration with ½ per cent novocaine), for at the previous operations performed under general anesthesia we were unable to elicit the snap on the operating table and so actually were unable to visualize the structures at fault. At operation the anterior border of the gluteus maximus was seen to be the offending structure. This was divided transversely for a distance of 1½ in., which eliminated the snap. The wound was closed but no plaster was applied. The patient was out of bed on the fourth day and on the seventh was walking without any snap; he was then discharged.

On June 7 he returned to hospital because of a reappearance of the snapping of the left hip. Operation was performed under local anesthesia on June 8. At operation the fascia lata seemed like a tight band which snapped across the trochanter. The fascia lata and the anterior edge of the gluteus maximus were both divided

transversely for a distance of 1½ inches, which relieved the tension and eliminated the snap. No plaster cast was applied. The patient was out of bed on the sixth and home on the ninth postoperative day. At the time of discharge from the hospital, there was no snap in either hip. One month later the case was followed up and neither hip snapped. Six months after the final operation he was again examined. At this time, there was a very rare and slight snap in the left hip region. This was not disabling so further operative treatment was not advised.

CONCLUSIONS

1. True snapping hip is a relatively unusual condition; bilateral snapping hip is rare.
2. The factors at fault may be a thickening of the iliotibial band or the gluteus maximus.
3. Operations for relief of this condition should be performed under local anesthesia. Early mobilization of the joint is essential.

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TYPHOID FEVER GALLSTONES*

THE relation between typhoid fever and gallstones is of more than clinical interest; it is of definite importance in the spread of typhoid fever.

We know that typhoid bacilli are eliminated from the body of a typhoid patient through the urine and feces. The Bacillus typhosus has been recovered with such constancy from the gall bladder on autopsy of patients dying of typhoid fever and from bile removed by duodenal drainage from living patients, that there is little doubt that the organism finds its way into the biliary passages in nearly all cases of typhoid fever and develops there. About 10 per cent of persons recovering from typhoid fever become carriers. The chronic carrier usually harbors the organisms in the gall bladder from which they are eliminated by way of the intestinal tract.

In view of the knowledge that typhoid bacilli so commonly lodge at least temporarily in the gall bladder, the theory might be advanced that the organism was the sole cause of gall-bladder disease in many instances. There are other factors, however. From a bacteriological study of the bile of cases coming to operation for gall-bladder disease, it has been found that 11 per cent are of typhoid origin while the cultures were sterile in 52 per cent. Typhoid organisms are found in the centre of gallstones quite often. The incidence of gallstones is variously given. They are found in about 6½ per cent of all persons coming to autopsy; they have been noted in about 12½ per cent of all persons coming to operation for some abdominal condition other than gall-bladder disease. Infection is recognized as the most important factor in causing gallstones and, of the organisms found, the typhoid bacillus heads the list.

An attack of typhoid fever may result in later gall-bladder diseases with possible stone formation. Also we see that the biliary tract harbors organisms for long periods of time after an attack of typhoid fever and that these so-called carriers may be a source of danger to others.

CASE REPORT

Mrs. M. S., aged forty-six, first seen January 9, 1928, at her home, complained of pain in the upper abdomen and vomiting. She stated that she had been taken two days previously with pain in the upper abdomen which felt like a tight cord just under the free margin of the ribs. The pain was not colicky. Following the onset of pain she vomited. The pain continued but she did not vomit again. At the time of

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examination, the patient had a temperature of 101°, a pulse of 92 and a respiratory rate of 24. There were a few moist râles at both bases posteriorly. There were moderate tenderness over the gall-bladder region and slight spasm of the right rectus. The patient stated that she had had similar attacks frequently in her early years but for the twenty years preceding 1926 she had been free from them. In 1926 she suffered a very severe similar attack. In 1914 she had typhoid fever and was moderately ill.

The next day the patient was admitted to the hospital. Upon admission, the temperature was 105°, pulse 120, respirations 28. Slight jaundice was noted. The chest still showed a few râles. The abdominal signs were essentially as before. The white blood count was 10,120. The urine showed sugar and albumin, acetone and diacetic acid. The blood pressure was 130–80. The patient looked sick and was sick. Digitalis was started and glucose solution given by rectum.

On the day following the patient was improved. The maximum temperature was 101.8°, pulse 94, respirations 22. Jaundice appeared about the same. The blood pressure was 110–66. There was no change in the chest findings. There was a definitely palpable mass in the gall-bladder region with moderate tenderness. The white count was 10,600; the urine showed a trace of sugar, a positive acetone and a negative diacetic reaction. The blood sugar was 150 mg. Widal's reaction was positive.

On the next day the patient was much improved. The highest temperature recorded was 100.2°. There was less icterus. The chest was negative. The abdominal signs remained unchanged. The Widal reaction was positive. The urine showed traces of sugar, acetone and diacetic acid. The Vandenburgh test showed an immediate positive reaction.

On the following day there was no visible jaundice. The temperature remained normal and the patient felt good. There were no signs in the chest. The abdomen showed a definite, tender mass in the gall-bladder region. The blood pressure was 132–78. The urine showed a faint trace of acetone and much bile. The coagulation time of the blood was two and one-half minutes.

On the next day the patient was operated upon and the gall bladder and appendix removed. The patient stood the operation well. A rubber tube was tied to the stump of the cystic duct to take care of any leakage. The gall bladder was transferred to the laboratory under aseptic condition where cultures made from the bile showed bacillus typhosus. The gall bladder was filled with stones. The laboratory report was chronic cholecystitis with cholelithiasis and chronic appendicitis.

The day after operation the patient was in good condition. She tolerated liquids. The urine was negative except for a trace of acetone. Two days after operation, a small amount of bile came out through the drainage tube. This continued for several days, presumably from the raw surface of the liver which I had been unable to peritonize satisfactorily. The tube came out on the seventh day after operation and the wound promptly healed. The Widal reaction at this time was positive. The patient made an uneventful surgical recovery but on January 23, nine days after operation, she showed a stool for positive typhoid organisms after previous ones had been negative. However, on January 28, 29 and 31, we obtained negative stools. The patient was discharged on February 4 in good condition.

This case presented several problems. When I first saw the patient, in spite of her history I was suspicious of a central pneumonia with referred abdominal symptoms. When the patient's temperature rose to 105°, I felt more inclined toward a pneumonia, for certainly gall-bladder cases rarely do this. With the urine report showing sugar and albumin, acetone and diacetic acid, I was further confused. Of course the persistant low white cell count was against a pneumonia. With the appearance of a palpable mass and clinical jaundice I felt more certain of the diagnosis. The changes in the urine are illustrative of the disordered chemistry of the body brought about by vomiting, limited fluid intake and pancreatic disturbance. The Widal test taken on speculation remained positive throughout, apparently due to her old typhoid condition.

In view of the fact that this patient had gallstone attacks before the typhoid fever, one cannot attribute the gallstone formation to the entrance of the typhoid bacilli into the biliary tract. Unfortunately no stool examinations were made before operation. They would undoubtedly have shown

typhoid organisms just as they did postoperatively. The typhoid bacilli had apparently been lodged in the biliary tract since the patient had typhoid fever. This made her a chronic carrier and it seems likely that she was responsible for new cases every now and then. Thus by surgical means she was relieved of a condition uncomfortable to herself and probably a menace to others.

BILATERAL ANEURYSM OF THE RENAL ARTERY*

William James Carson, M.D.

MILWAUKEE

DANIEL NEBEL[1] in 1717 first described aneurysm of the renal artery. Keen[2] in 1900 collected 12 cases from the literature and reported a case in which he performed a right nephrectomy. Orth[3] in 1919 was able to collect 31 cases from the literature and reported 1 of his own. Since the appearance of Orth's article, 16 cases have been reported by Vogeler[4] (not confirmed by operation or autopsy), Conroy,[5] Roth,[6] Rowlands,[7] Schramm,[8] Richardson,[9] Soderlund,[10] Healey,[11] Chisholm,[12] Renck,[13] (2 cases), Callahan and Schiltz,[14] Dorndorf,[15] Meyer and Singer,[16] Weiss,[17] and Singer,[18] to which may be added 1 case described below, bringing the total to 49.

CASE REPORT

F. V., white, male, aged sixty-two, was first seen on October 6, 1925, in the dispensary, complaining of frequency of urination (hourly during day, night, two to three) hematuria, and painful nodules over the abdomen and back which had been present for years. A diagnosis of carcinoma of the bladder, adenoma of the prostate (cystoscopic) and von Recklinghausen's disease was made. As the patient refused hospitalization, deep roentgen ray treatment, one and one-half hours each day for four days, was given. On November 6, 1925, he was admitted to the University of Maryland Hospital, complaining of severe pain over the bladder and profuse hematuria. He died November 7, 1925.

Laboratory findings: November 6. Urine: sp. gr. 1.012, albumen 2 +, sugar negative, large number of W.B.C. and R.B.C. Blood: R.B.C. 3,800,000; W.B.C. 13,200; hemoglobin 60 per cent, blood chemistry: N.P.N. 0.37 mg., sugar 0.118 mg. per 100 c.c. blood. Wassermann reaction negative.

Anatomical diagnosis: carcinoma of the bladder involving the base and lateral wall, obstructing the right ureteral orifice; metastases to the lymph nodes along the internal iliac arteries, abdominal aorta, and spleen; hydroureter, right; hydronephrosis, right; ureteritis, bilateral; adenoma of prostate, all lobes; aneurysm of renal artery, bilateral; von Recklinghausen's disease; solitary cyst, left kidney, upper pole;[19] two large cysts on anterior surface of right kidney.

Right renal artery divides into three branches at a point 3 cm. from the aorta. On the anterior surface of the artery, proximal to its division into branches, a definite dilatation measuring 10 by 14 mm. is seen. This dilatation is a sacular aneurysm, its wall measuring 2 mm. in thickness. The diameter of the aneurysm is 14 mm. The intima is smooth and glistening and in direct continuity with that of the artery.

Left renal artery divides into three branches 3.5 cm. from the aorta. Between the bifurcation of the superior and middle branches a dilatation measuring 8 by 6 mm. is seen. On section this is seen to be a definite aneurysmal sac. The wall is in direct continuity with that of the artery.

Microscopical Examination. Sections taken from proximal part of renal arteries show moderate thickening of the tunica intima, with the elastic lamina well preserved. Sections through the sac show the wall to be composed

* From the Department of Pathology, University of Maryland. Read before the Chicago Urological Society, May 24, 1928.

of fibrous connective tissue for the most part, with areas stained as a pale pink homogenous mass. Few muscle fibers are seen, and no elastic lamina can be made out. The intima is poorly stained.

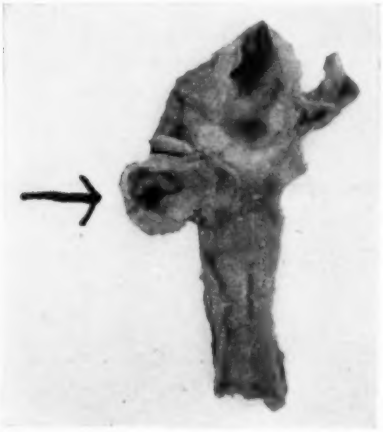

Fig. 1. Right renal artery showing sacular aneurysm 14 mm. in diameter.

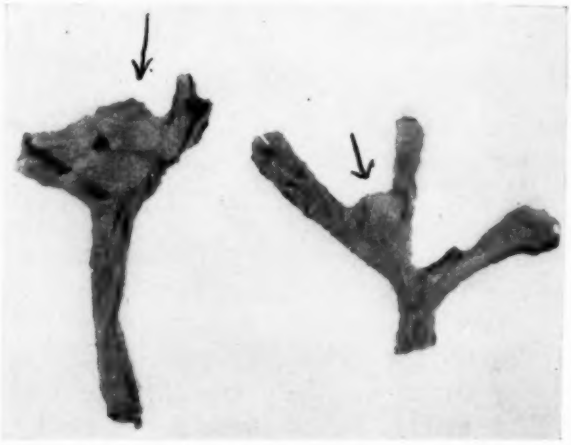

Fig. 2. Left renal artery showing aneurysm between the bifurcation of the superior and middle branches.

INCIDENCE

The incidence of this condition is very small as shown by Mueller.[20] In a study of 6425 autopsies he found 171 aneurysms of which only 3 were located in the renal artery. Bosdorff[21] found 93 aneurysms in 3108 sections, not one of them being renal. Emmerich[22] found 1 renal aneurysm in 7669 autopsies. In a recent study of the renal blood vessels in 400 bodies no other case was encountered.

Age Incidence. In the cases of traumatic origin, this varies from fifteen to fifty-six years; average, thirty-six. In the spontaneous cases the ages vary from nine to eighty-two years with a general average of fifty-six. The average age incidence for all cases of both types in which an age was given is forty-six years.

ETIOLOGY

In the etiology of renal aneurysm, trauma is of first importance. Conroy found 17 cases due to trauma, 6 cases associated with generalized arteriosclerosis, and 9 cases occurring in patients who had had severe infections.

TYPES

All aneurysms have walls continuous with the walls of the blood vessel. The literature on renal aneurysm contains 18 cases classified as "false" representing retroperitoneal, perirenal or the accumulation of blood or blood clots, which are the result of leakage from the renal vessel. They are usually located at the bifurcation of the renal artery or on its various branches, varying in size from 4 mm. to 5 cm. in diameter.

SYMPTOMS

The combination of abdominal pain, followed within a short period of time by the passage of bloody urine, is the most characteristic feature about the onset of the illness. In the majority of cases it is associated with trauma. A stitch-like pain over the kidney, which occurs in infarcts of the kidney, was described by Weiss. Hematuria was profuse enough to exsanguinate the patient in all instances, and intermittent hematuria has been reported in 9 cases. Dysuria may result from blood-clot filling the urethral orifice.

PROGNOSIS

Renal aneurysm which produces symptoms, is fatal in 100 per cent of cases where there is no surgical intervention. Usually there are severe attacks of pain and hematuria over a period of weeks or

months, and death results from hemorrhage into the renal pelvis, retroperitoneal tissue, or peritoneal cavity. The longest interval of time between onset of symptoms and death was five years (Morris.[23])

In the 15 operations performed, up to the present time 13 patients have been cured and 2 have died. Nephrectomies were done in 13 cases.

Orth was able to turn out the clots and successfully suture the tear. Callahan excised the sac and preserved the artery. Orth reported good function one year later and Callahan six months after operation.

TREATMENT

For those cases which give rise to symptoms removal of the sac together with the kidney is the best procedure.

CONCLUSIONS

Aneurysm of the renal artery is rare. The cause of the condition can be divided into (1) traumatic, (2) primary disease of the arterial wall, either degenerative or inflammatory and (3) secondary disease due to extravascular morbid processes.

The forty-ninth case of true aneurysm of the renal artery is reported.

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EDITORIAL

LOCAL TRAUMA AS A CAUSATIVE FACTOR IN CANCER

THAT a single local injury is not infrequently the direct or exciting cause of malignant tumors of nearly all types, has been very generally believed by medical men who have had a large clinical experience with cancer. This view is even more deeply rooted among the laity by whom it has been held for many centuries. It has been frequently disputed, however, by high authorities, chiefly pathologists who, in spite of their great knowledge of the histology and pathology of tumors, rarely have the opportunity of studying the disease in the living human being and still more rarely see it in its early beginnings. On the other hand the clinician, the man who is first consulted in a case of malignant tumor and who at once obtains a careful history of the case, should be more capable of giving a judicial opinion as to the value of the evidence bearing upon the question of trauma. With so many high authorities on record as doubting or denying the possibility of a local injury producing a malignant tumor, the clinician has often hesitated to express his own opinion to the contrary, convinced though he may be of its truth. During recent years, with greater care in the recording of clinical histories, one cannot fail to be impressed by the large and increasing proportion of cases of both sarcoma and carcinoma in which there is definite evidence of a local injury occurring a few weeks or months prior to the appearance of the tumor. While many pathologists and some clinicians still refuse to accept the seemingly convincing evidence of a causal relationship simply because they can find no scientific explanation of it, the workmen's compensation commissions and higher courts both here and in Europe have ruled that local injury may be a competent exciting cause of cancer. In rendering such decisions they have brought to bear upon the question the same logic and common sense that have enabled them to settle other complicated and difficult problems that have come before them. Nowhere is this shown more clearly than in

a recent decision of the Supreme Court of Appeals of Virginia, quoted by Stephens in The American Journal of Surgery for October, 1928:

The Supreme Court of Appeals of Virginia, in affirming an award of compensation under the workmen's compensation act for the death of an employee from sarcoma after an injury to his right side, says that whatever view the court might take of the medical opinions, they were frankly and at best but theories . . .

To this the court may add that the courts have in general found no difficulty, in cases similar to this one, in applying the ordinary rules of evidence, and in drawing the ordinary conclusions of cause and effect from established facts, and this court finds none. This, the court doubts not, courts will continue to do with a full sense of justification and without apology until the cause of cancer is definitely and scientifically established.

In the last two issues of The American Journal of Surgery there have appeared two papers bearing upon this subject. In the first, "Bone Sarcoma," by Stephens[1] the writer attempts to show that local trauma as an exciting cause of cancer has but little evidence to support it. In the second paper, "Trauma, Carcinoma and Workmen's Compensation"[2] the author, Wainwright, contends that abundant proof has already been offered to show that local trauma may be unquestionably the exciting cause of the development of carcinoma. These two papers well illustrate the widely divergent opinions which are held at present among the profession at large. The reason for this great difference of opinion, we believe, is, that few of the average surgeons or physicians find time to make a study of the question from first hand data. They get their opinions from other and often early writers and, if their own experience does not support the view expressed, they lack the courage to dispute it.

Stephens, in his paper already referred to, states: "Our literature is filled with reports of clinical and pathological investigations dealing with the question: Lowenthal in 1875; Gross in 1897; Coley in 1898." Every one of these authors upholds the view that antecedent local trauma is a competent producing cause of both sarcoma and carcinoma. One cannot help but ask the question why Stephens did not quote the later statistics of the authors already mentioned, which would either offer further support of the theory or discredit it entirely. My first paper on the subject published in 1898 was based upon a study of 170 cases of sarcoma in which a history of antecedent trauma was noted in 46 cases or 27 per cent.[1] At the time of writing my opinion was entirely unbiased, but since the publication of that paper I became deeply impressed with the large number of cases of sarcoma giving a definite history of local trauma at the site where the tumor later developed, so that thereafter, in every case of malignant tumor, I made a special effort to inquire most carefully into the early history as regards the presence or absence of antecedent local injury, and if present, to ascertain the exact nature and severity of the injury. My later experience based upon a far more comprehensive study of the question entirely confirms my earlier conclusions and the evidence now accumulated is so strong that I no longer have the slightest doubt that both sarcoma and carcinoma may be caused by a single local trauma.

In my paper on "Injury as a Causative Factor in Cancer[2] I stated:

The question of the causal relationship between trauma and cancer should be determined by a careful scientific and judicial study of all the facts bearing on such relationship. That is a question entirely independent of our ability or inability to offer a satisfactory explanation as to the nature of such relationship.

Both Phelps and Segond make too great an effort to discredit the direct statements of intelligent patients. When a woman of more than ordinary intelligence strikes her breast

[1] Stephens, Philip. 5: 364, 1928.
[2] Wainwright, J. M. 5: 433, 1928.

[1] *Ann. Surg.*, March, 1898.
[2] *Ann. Surg.*, 53: 1911.

against a sharp corner of a bureau, causing the characteristic signs of a local contusion (ecchymosis and tenderness); when a careful examination of the place immediately after the injury fails to reveal the presence of any tumor, but one or two or three weeks, or a month later, a hard tumor develops at exactly the point of injury, the supposed causal relationship may, with Phelps, be attributed to the "unreliability of patients' logical processes, which have hypnotized the attending physician into accepting the improbable assertions as undoubted facts." But when, in the case of a fractured humerus in a man in perfect health, we have an x-ray photograph taken immediately after the fracture, as in my case, showing absolutely normal bone structure and a few weeks later another photograph shows a typical sarcomatous tumor developing at the exact site of the fracture, and when subsequent operation with microscopic examination prove the correctness of the diagnosis; and again, when a surgeon makes an incision in the inguinal region for the operation for inguinal hernia, through absolutely normal structures, and four weeks later there develops at the exact site of the incision, involving all the layers of the scar, a rapidly growing round-cell sarcoma[1]—we have facts which in no way depend upon the logical or illogical processes of patients, and the explanation of which demands something better and more profound.

Stephens quotes Bloodgood as saying that in 1000 bone tumors of which about 400 were sarcoma he saw only 2 cases in which a roentgenogram was taken directly after the injury. While we in no way admit that it is necessary to have a roentgenogram taken immediately after the injury to prove that a sarcoma developing some two months subsequently was not already present at the time of the injury, my own statistics furnish a considerable number of cases in which such roentgen-ray examination was made and found entirely negative. I can recall 6 cases in which the roentgenogram proved negative and yet within a few weeks or months a highly malignant tumor developed at the exact site of the injury. In our opinion, the following case without an immediate roentgenogram is quite as conclusive evidence:

W. B., male, aged twenty-eight, in the middle of September, 1925, was struck a severe blow on the occipital region by a heavy wooden packing case which fell from the top of a nearby pile. A hematoma the size of half a hen's egg developed immediately at the site of the injury. Under local applications this disappeared within two weeks' time. It then reappeared and began to increase steadily in size until by October 2, it had become larger than it was originally. A diagnosis of hematoma was made by Dr. Walter Jones, the surgeon for the insurance company. An incision was made following which there was profuse hemorrhage. The patient was sent to the Hospital for Ruptured and Crippled, under the care of Dr. J. P. Hoguet. Physical examination showed a large swelling in the upper occipital region, the size of half an orange, 3½ in. in diameter, symmetrical in shape, and fluctuating. Roentgen-ray examination and exploratory incision showed extensive destruction of both tables of the skull at the site of the swelling. Under radium-pack treatment and long-continued toxins, the patient made a complete recovery and is in excellent condition at present, more than three years later. Microscopical diagnosis by Dr. James Ewing: "Small spindle-cell sarcoma, very vascular; no bone formation, probably an osteogenic tumor arising from periosteum."

To assume that this man had a preexisting tumor of the skull at the exact site of the injury, without any previous symptoms, a tumor which grew with such rapidity that it destroyed both tables of the skull in three or four weeks, is neither scientific nor according to common sense.

While Stephens has no new facts to prove that local trauma cannot be an exciting factor in the development of malignant disease, Wainwright, on the other hand, from his own experience contributes new proof that it does and this, in the field of carcinoma wherein it has always been admitted that local trauma plays a much less important

[1] In this patient there was a primary but hitherto undiscovered sarcoma of the jaw and the local trauma incident to the hernia operation, by lowering the resistance of the tissues, produced a favorable nidus for the causative agent, be it sarcoma cell or some microorganism, to obtain a foothold and denelop.

etiological rôle than in sarcoma. In this connection, the following personally observed case may be of interest:

L. F., male, aged forty-nine, had always been well until May, 1908, when he was struck over the right malar region by a steel lever (patient worked on the railroad). A swelling appeared immediately and he consulted a physician on the same day. Poultices were applied for the next two weeks. The swelling continued to increase in size gradually and finally became ulcerated over the central portion. Physical examination on June 5, 1908, a little over three weeks after the injury, showed a man of strong physique, in perfect general health. On the right side of the face, in the malar region, was a tumor the size of a goose egg, movable upon the deep parts, apparently originating in the muscle and fascia. The skin was very much reddened and in the center there was an ulcerated area about 1½ in. in diameter; no enlarged glands. On removing the dressing there was profuse hemorrhage.

Although the condition seemed practically inoperable, I attempted to remove the growth, if possible, and operated on the following day. It was impossible to remove the entire tumor and a portion had to be left behind. The patient was immediately put upon toxin treatment. In July it was possible to remove the remaining portion of the tumor and this was done by my associate, Dr. William A. Downes. In spite of these operations and the toxin treatment, the tumor very quickly recurred, and the patient's general health soon became affected. The treatment was discontinued. The disease progressed with great rapidity and caused death within three months from the time of the injury.

In this case a microscopical examination was made by Dr. W. C. Clark and Dr. James Ewing both of whom pronounced the disease to be carcinoma.

In 1926, in a paper on "Local Injury as a Causative Factor in Bone Sarcoma with Especial Reference to the Medico-Legal Aspects[1] I gave an analysis of 360 cases of sarcoma of the long bones personally observed over a period of thirty-six years, or from 1890 to 1926. In 181 cases or 50 per cent. there was a definite history of antecedent local trauma. In 133 cases or in 70 per cent, the tumor was noticed within the first six months after the injury and in 30 per cent, it developed within the first month.

This paper contains numerous examples of so-called acute traumatic malignancy in which the sarcoma developed at the exact site of the injury within a very short time (two or three weeks or less) after the injury and in which there was no evidence pointing to even a suspicion of any abnormal condition prior to the injury. It is only fair to state that there are on record a few cases in which a pathological fracture has occurred following a very slight injury and in which a roentgenogram at the time or shortly after showed a primary sarcoma of bone or a secondary carcinoma. Two such cases have come under my own observation, in both of which an attempt was made to secure compensation, in 1 case on the ground that the injury aggravated the pre-existing condition (malignant tumor) and in the other case an attempt was made to ignore the pre-existing tumor even though the roentgenogram showed evidence that it was of long duration and not the result of the injury. These cases are extremely rare, however, and have no real bearing on the question at issue.

We cannot agree with Stephens in his statement that: "until the actual cause of the disease (cancer or malignant tumor) is discovered we cannot hope to get a clear understanding as to what effect, if any, trauma has on its development."

While medical men may be willing to wait until the cause of cancer is discovered before admitting that local injury may be a causative factor in the development of malignant tumors, it is quite evident that the workmen's compensation commissions and the higher courts are not willing to await this uncertain and possibly remote date. Furthermore, we believe the latter are quite right in the statement that most of the medical opinions on the subject at present are but theories. Such being the

[1] *Internat. J. Med. & Surg.*, July & Aug., 1926.

case, they have very wisely applied the plain rules of evidence and common sense and reached a conclusion that is not only logical but in accord with the facts. An analysis of a very large number of case reports of bone sarcoma personally observed confirms this opinion. To refuse to accept as a fact something based upon experience and supported not only by a single observation but a great number of observations, simply because we are not at present able to give a complete and scientific explanation of it is not only unscientific but illogical. As has been stated recently by Dr. Robert A. Millikan, one of the most distinguished men in the world of science today: "a great body of definite and certain knowledge can be safely and securely built up on the basis of a few fundamental empirical facts even before we have any fundamental comprehension of the nature of the causes of these facts."

We might as well have said a decade ago that we would not use quinine in the treatment of malaria because not knowing the cause of the disease we had no satisfactory explanation of why this drug should be efficacious. Reasoning in the same manner, we would have refused to use cod-liver oil in the treatment of rickets because, at the time, we knew nothing about vitamins and had no actual explanation of why it should be of benefit.

As regards the medico-legal aspects of the question, while we believe it has been proven beyond doubt that a single local injury may be an important causative factor in the development of both sarcoma and carcinoma, this does not mean that in every case of sarcoma or carcinoma with a history of antecedent injury, more or less indefinite as to location and still more indefinite as to date of occurrence, the injury should be regarded as sufficient to establish a causal relationship. It is only fair that the interests of the insurance companies should be safe-guarded as well as those of the patient; and in my opinion, compensation should be restricted to those cases which fulfill the conditions so admirably laid down by Segond,[1] as follows:

1. The authenticity of the trauma.
2. Sufficient importance or severity of the trauma.
3. Reasonable evidence of the integrity of the part prior to the injury.
4. Correspondence of the tumor to the site of the injury.
5. A date of appearance of the tumor not too remote from the time of the accident to be reasonably associated with it.
6. A diagnosis established by clinical and x-ray evidence, supported when possible by microscopical confirmation.

William B. Coley.

[1] Segond. *Trans. Cong. French Surgeons*, October, 1907.

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BOLOGNA, ITALY

PART I

IN Goethe's "Goetz von Berlichingen," the monk Martin asks Godfrey's name and the warrior offers him his left hand: "Warum," asks the offended Martin, "reicht Ihr mir die Linke? Bin ich die ritterliche Rechte nicht Werth?"[1]

Godfrey answers: "Und wenn Ihr der Kaiser wärt, Ihr müsstet mit dieser vorlieb nehmen. Meine Rechte, obgleich im Kriege nicht unbrauchbar, ist gegen den Druck der Liebe unenpfindlich; sie ist Eins mit Ihren Handschuh; Ihr seht, er ist Eisen."[2]

Then Martin suddenly stoops to kiss this iron hand which reveals to him the name and heroism of the famous captain.

Thus in Goethe's poem is immortalised the historic artificial limb, which served as substitute for the hand that Goetz von Berlichingen (1480–1562) (Fig. 1) lost in his twenty-third year in the siege of Landshut in Bavaria, and which to quote Goetz' own words: "In Gefekt mehr Dienste geleistet hat als zuvor die Natürliche."[3]

This apparatus, which according to some was the work of an artisan of Nuremberg, according to others of Goetz' own armorer, who lived in Holnhausen, near Jagsthausen, represents the most striking example of antique artificial limbs.

Fate has allowed this curiosity to survive in duplicate, one of a rougher, simpler pattern, possibly the first which the knight used and which is now in the possession, as an heirloom, of the family of Berlichingen-Rossach; the other, an admirable triumph of mechanics, is preserved in the knight's native castle near Jagsthausen (Fig. 2).

A book is placed beside the hand for recording the impressions of visitors. Kotzebue wrote in it: "This hand which fought only for the right, was governed by a heart which beat only for virtue. The hand remains empty . . . Nature, Nature, why hast thou destroyed the heart!" Brockmann, the Viennese actor wrote: "In fiercer times men built hands of iron to fight for liberty; in gentler days weapons and chains to suppress liberty."

There exist numerous copies of this hand of Goetz; one, constructed for the Emperor Joseph II, is in the Museum at Vienna; another is in the armory of Bredow of Wagnitz (Mannheim), and a third forms part of the famous collection of Meyrik in Wales.

But although the hand of Goetz is the most famous, it is by no means the only example of the artificial limbs of past centuries. One which is supposed to have been constructed in the fifteenth century, but which we think to be of later date, is in the Imperial Museum at Berlin. Unlike the hand of Goetz, this one has mobility in the metacarpophalangeal joint of the thumb,

* Translated by M. Forrester Brown, M.D.

while the forearm is connected to the arm by a hinge like the scales of a fish. Another artificial hand was fished up in 1836 in the

Fig. 1. Goetz von Berlichingen.

Rhine canal near Alt-Ruppin and is now in the Neu-Ruppin Museum. It has a thumb moving at the metacarpophalangeal joint and fingers jointed in pairs. It seems to us of earlier construction than the Berlin hand. A third is in the private museum of Count Hans Wilczel of Vienna and is said to be of French origin. The thumb is fixed and the fingers move in pairs. We remember seeing several wooden hands of the seventeenth century in the Deutches Museum in Nuremberg.

L. Raymond, quoted by Rivière, describes a mosaic in the cathedral of Lescar (Lower Pyrenees) which according to some authorities (Longpérier) belongs to the Gallo-Roman epoch, according to others (Raymond) to the twelfth century and in which is portrayed a negro whose left leg, lacking the foot, takes its support from the knee from the socket of a wooden pilon, exactly similar to those in use at the present day. Rivière also describes the counter drawing of a fragment of vase found in Paris in 1862 in which is shown, among other figures, a man seated in a chair with a high back, whose right leg, amputated about the seat of election, is attached to a pilon with a forked end.

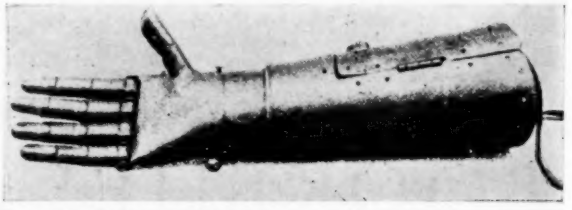

Fig. 2. The hand of Goetz von Berlichingen (second model).

It is clear that, rather than true artificial limbs, in the modern sense of the word, these pictures represent rough apparatus, which now-a-days, as then, the poorer classes of maimed and crippled are in the habit of making for themselves and which are portrayed even more clearly than in prehistoric vases and antique mosaics, in the engravings and pictures of Cornelius Mastys, Gerolamo Bosch and Brughel and in the frescoes of Orcagna in the Camposanto at Pisa and those of Penni in the Vatican.

Herodotus (484 B.C.) records that Egesistratus, after being taken prisoner and chained up, escaped by freeing himself through amputating the foot that was chained. When the wound healed, he provided himself with a wooden leg. In the Talmud there are references to pilons and artificial hands. Pliny the Younger (Book 7, Ch. 29) notes that M. Sergius, after losing his hand in the second Punic war; "dexteram sibi ferream fecit, eaque religata proeliatus Cremonam obsidionem exeruit."

My researches have not enabled me to ascertain the existence of other artificial limbs than those enumerated above. Indeed, information is scarce about them in history. It is easy to understand how the origin of artificial limbs reaches less far back than that of surgical instruments which, as is well known, date back to prehistoric times. The artificial limb is complementary to a surgical amputation; which must not kill the patient by hemorrhage, but produce a useful stump and such an amputa-

tion can only be obtained after the discovery of some method (cautery) of checking loss of blood from the vessels. Prior to that, amputation could only consist in the destruction of segments of a limb by crushing, by which method the stumps obtained were unsuited to any type of artificial limb.[4] It is certain that the lower limb, being both more needful and easier to construct, will have preceded the construction of the upper limb.

There is no lack of iconographic illustrations and very early records of the use of artificial limbs.

On an Italian vase of the fourth century B.C., belonging to the Louvre, and described by Longpérier and Rivière[5] there is represented a cripple supporting himself on a stick, whose lower part forms a pilon. This is less a limb proper than a walking stick which is, so to speak, fused with the deformity. Rivière dilates at length on the deformity from which this comic figure suffers, but he fails to classify it. Charcot and Richer, who repeat Rivière's description, do not commit themselves. It does not seem to us, as Rivière believes possible, that it is a case of congenital deformity. This could only be congenital flexion of the knee; but then the knee would not have the shape portrayed by the artist, and the leg would be differently rotated. We think that this most probably represents a deformity from anterior poliomyelitis. The thigh is much rotated outward and the leg flexed on the thigh. The right foot is covered by the scrotum, the left, to which none of the authorities have paid attention, is in extreme equinus. Such rotation and a flexion deformity of this degree are not rare among the deformities of neglected poliomyelitis. The equinus of the left foot seems to confirm this hypothesis.

Guido Guidi, the Florentine surgeon who practised so successfully at the court of Francis I of France, where he became friends with Benvenuto Cellini, writes in Chapter 7, Book IV in his works collected by his nephew, who had the same name: "Excisa manu, ferrea alia brachii alligatur, quae usum aliquem ad praebendum et continendum prestat. Exciso crure, sunt qui ligneum parent, quod non solum ad decorem, sed etiam ambulandum accomodatur."

Giovanni Tommaso Minadoi, a surgeon of Rovigo at the end of 1500 who, as medical attendant of the Venetian consuls, travelled widely in the Orient and wrote a history of the war between the Prussians and Turks (1576–88), was doctor to Guglielmo Gonzaga, Duke of Mantua and professor in the University of Padua, asserts that he observed two amputation cases of which one, after the loss of both hands, could by means of apparatus (iron) take his hat off and on, open and shut a purse and sign his name; the other, after the loss of his right hand, could do many acts.[6]

Paolo Giovio relates that the Turkish corsair Horuk, surnamed Barbarossa, lost his right hand in the battle of Bugia (1517) against the Spaniards and had an iron one made "qua ad cubitum religata multis in proeliis postea feliciter usus est."

Duke Christian of Brunswick, who had lost his left hand at the battle of Fleury (1622) had an iron arm made by a Dutch workman.

An artificial hand which has acquired historic importance, because it was depicted and described by Ambroise Paré, is one constructed in Paris in the middle of the sixteenth century by a master smith, and which is called "le petit Lorrain." (Fig. 3.) The hand, which on the dorsum has the form of a steel gauntlet, is attached to the forearm by two metal rods and leather straps. The thumb is rigid and the fingers are kept extended by four springs fixed in the palm. When they are flexed, they are kept so by rachets worked by metal levers. Paré also portrays another upper limb in which the mechanism which controls flexion and extension of the elbow is clearly shown and easy to understand.

We will not linger further in an analysis of apparatus in the centuries before the renaissance of surgery, for our aim is simpler. What has been described is only a

succinct introduction to the description of certain ancient artificial limbs, which are worth studying and will be described.

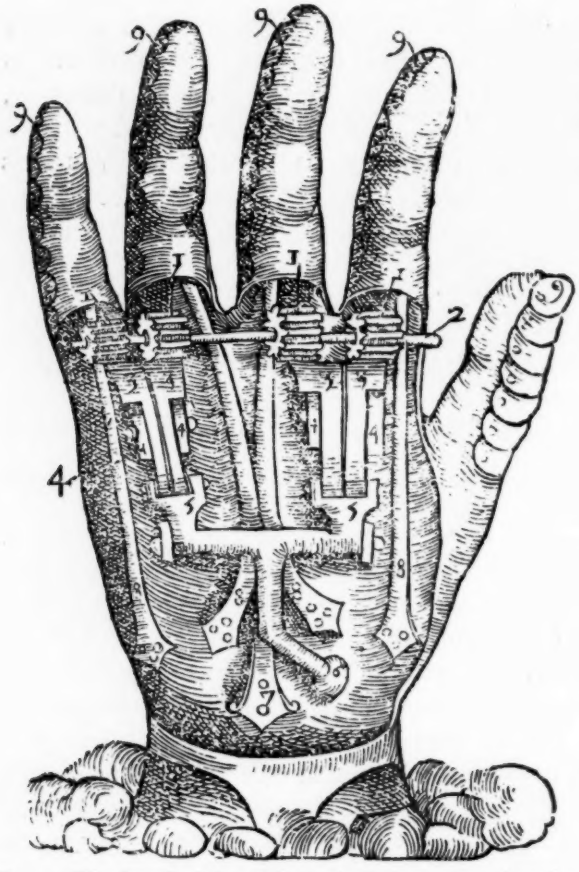

Fig. 3. The hand of "petit Lorrain," demonstrating the mechanism.

Some time ago Dr. Capparoni, noted as a student of medical history, informed me that there existed in the Museo Stibbert[7] in Florence some antique limbs, which he thought worthy of study.

On the recommendation of Ugo Ojetti and thanks to the extreme kindness of Cav. Alfredo Lensi, director of the museum, I have been permitted at my leisure to examine, photograph and draw these limbs collected in the Museum.[8]

By chance, while recently visiting the Museo Poldi-Pezzoli in Milan, I discovered in the Sala delle Armi, an artificial upper limb which has many resemblances to that of Stibbert. This one also I have been kindly allowed to study and photograph through the courtesy of Comm. Giovanni Beltrami, director of the Museum.

The description of the Stibbert and

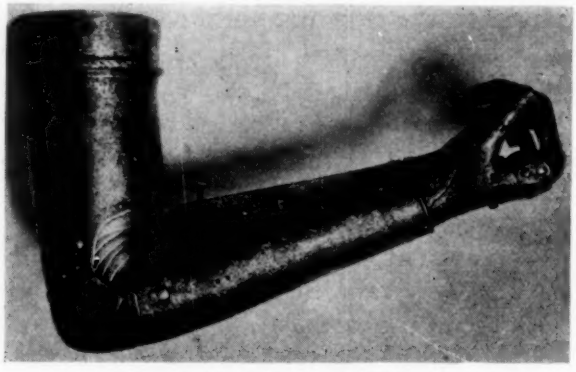

Fig. 4. Prosthesis A (Stibbert).

Poldi-Pezzoli limbs will be followed by that of a curious and original apparatus for deformity of the lower limb, constructed in the seventeenth century, which the engineer Dino Zucchini, its present proprietor, has allowed me to examine and describe.

PROSTHESIS A (STIBBERT)

No. 3819 Fifteenth and Sixteenth centuries.[9] Weight gr. 1330, (Fig. 4).

This prosthesis is of iron and is for a right arm stump. It consists of upper arm, forearm and hand. The opening of the armpiece is furnished with a ring, which by rotating round the long axis of the armpiece, allows small movements of rotation in the apparatus. This ring has a hollow corresponding to the axilla. The armpiece is a cylinder and is joined to the forearm-piece by three metal scales placed in the olecranon part of the apparatus, which are counterpoised in the sheath of the so-called *cubitiera* of the armor (Stibbert Catalogue, 1:188). The fold of the elbow has an anterior protection identical to that found in armor and which the Italian armorers call *alla moderna*.[10]

It is formed of numerous metal scales, overlapping so as to allow a fairly wide range of flexion and extension of the elbow, while protecting the flexure. In the apparatus under discussion there is a range

from 180° extension to 75° flexion. From full extension, the elbow can be flexed in three stages. This movement is controlled by a mechanism hidden in the interior and worked by a metal button projecting on the anterior surface of the forearm. From flexion, extension can be attained in three stages, or in one, by pressing on the button, noted above. The forearm-piece consists of two troughs of sheet-iron riveted together. The hand is in full supination and is attached to the wrist of the apparatus by three metal screws. It is formed of two parts, the carpometacarpal with which the thumb is continuous, and the digital part with the four fingers which are in semiflexion. The two parts are rivetted together. The rigid thumb is fixed in a position midway between abduction and adduction. The fingers are formed of a dorsal trough, which in its distal part is completed by a ventral trough. The fingers are kept open by a spring fixed on one side to the interodorsal aspect of the thumb and on the other side to the inner wall of the gutter for the fourth finger. Passive closure of the fingers occurs in three stages by means of a toothed lever, which locks in a transverse bridge at the root of the third finger. The lever is controlled by a long handle which projects on the palmar aspect of the wrist and it has its fulcrum on a metal bridge screwed to the inner aspect of the metacarpal trough.

There is only a small range for the fingers, whose pulps in maximum flexion do not touch the palm. The thumb does not touch the index and a space of about 1 cm. remains when they are nearest.

A series of holes all round the edge of the armpiece suggests that the proximal part of the apparatus, in contact with the stump, was completely covered in; but nothing remains to show how it was suspended from the stump or shoulder.

PROSTHESIS B (POLDI-PEZZOLI)

No. 945 in the Museo Poldi-Pezzoli inventory. Sixteenth century [?], (Fig. 5).

This apparatus is described in the inventory as: "Arm-piece in perforated sheet-iron, with gauntlet and artificial hand, also of iron."

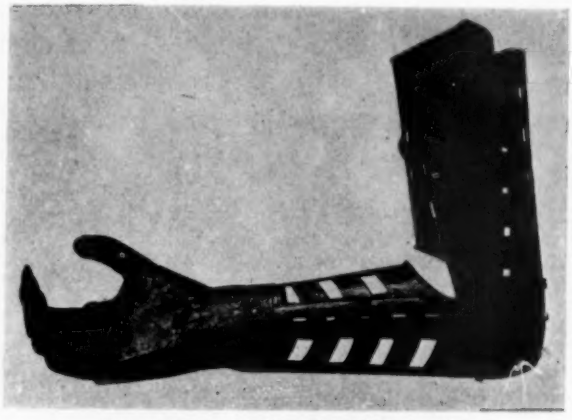

Fig. 5. Prosthesis B (Poldi-Pezzoli).

This prosthesis is of iron and is for the right arm stump. It consists of arm, forearm and hand. The armpiece consists of two troughs joined together by two medial hinges. A large buckle on the posterolateral aspect of the posterior trough, and which evidently corresponded to a strap, enabled the anterior trough to be closed and tightened on the posterior one, after the stump had got in. The upper part of the arm in front bears an iron rosette, elegantly chiselled and evidently placed there as an ornament, as in some armor. (Table 141, Stibbert Catalogue.)

The arm articulates with the elbowpiece, which in its turn articulates with the forearm. The respective joints are simply constructed with rivets (medial and lateral). The maximum flexion allowed the elbow is 80° and the maximum extension 160°. Ordinarily the elbow is rigid at right angles. To extend it two manoeuvres are necessary: (1) Extension of the arm on the elbowpiece by pressing on a button in the posterior aspect of the armpiece. This button controls a spring (placed within the armpiece) which when compressed withdraws a spur, this spur projecting against the front edge of the elbowpiece, prevents extension of the arm on the latter. (2) Extension of the forearm on the elbow, by an identical manoeuvre through press-

ing a button on the dorsal aspect of the forearm.

The forearm is cylindrical, formed of a

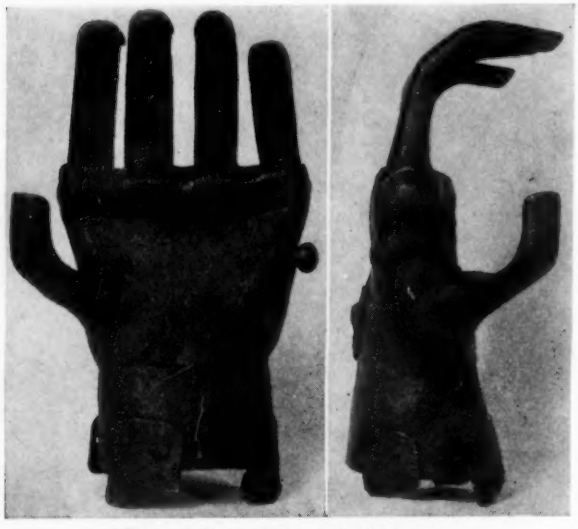

Fig. 6. Prosthesis c (Stibbert).

single sheet of iron with the margins united by rivets on the medial aspect. Both arm and forearm as fenestrated with rectangular openings arranged like fish scales (reminiscent of those in the Imperial Museum at Berlin) and with some, also rectangular, arranged in the longitudinal axis of the limb.

The hand with rigid wrist is nailed to the forearm in a position midway between pronation and supination. The carpo-metacarpal segment is formed of two pieces, dorsal and palmar, united by nails.

The thumb, rigid in the carpal and phalangeal joints is fixed to the metacarpal joint in abduction, while the second phalanx is semiflexed. The four fingers are united to the metacarpal part by a transverse bar, very oblique and supported in two holes through two ears which spring from the base.

The fingers are fixed to a transverse rod at equal distances from one another, and are semiflexed in the interphalangeal joints. The flexion is greater in the proximal than in the distal joint. The index is shorter than the middle finger, which is longer than the ring finger. The palmar aspect of the fingers and thumb is not cylindrical but levelled into flat surfaces. The nails are elegantly moulded. In its maximum closure the pulp of the fingers reaches to within 3 cm. of the wrist, while the fingers and thumb enclose a space roughly triangular with sides 3 cm. long. From full extension, the fingers can be carried into flexion in seven stages, thanks to a mechanism partly hidden within the metacarpal section, which consists essentially of a ratchet, forming part of the cylinder to which the fingers are fixed and also of two strips of spring-steel, one of them attached to the front wall of the palmar trough, controlled by a button projecting in the palm and articulating by its free end with the ratchet; the second spring, an antagonist of the first, is fixed to the inner wall of the dorsal trough and by its free end engages in a cavity cut in the cylinder referred to above. When the fingers are in full flexion, pressure on the palmar button releases the free end of the first spring from the ratchet, and allows its anatagonist to restore the cylinder to its original position, i.e. with extension of the fingers.

This apparatus has many advantages in construction superior to that last described: Physiological attitude of the forearm; good construction of the hand; correct balance of the grasping interval and irreversibility of the movement into flexion. But these qualities, which would be most valuable for a forearm stump, have little importance in an apparatus which is evidently intended for an arm stump. That this is so is proved by the presence of a mechanism for moving the elbow, a device admirable on its own account for the smallness of the space it occupies and thence for its adaptability to a case of longarm stump.

It is obvious that in the construction of this apparatus, the mechanic, who was certainly an armorer, has been guided by the aesthetic standard of armor. The apparatus, made as it is with the hand in

complete supination and the fingers fully flexed, could only be useful for passively holding the reins in riding.

PROSTHESIS C (STIBBERT)

No. 3316. Fifteenth century. Weight 530 gm. (Fig. 6).

This prosthesis is made of large sheets of metal and forms a left hand. It is indisputably a working hand. There is obvioms neglect of any attempt at aesthetic effect. The only accurate reproduction is in the wrinkles on the dorsum of the joints, which are indicated with much grace and truth, and the nail beds, which are fairly well done. Otherwise the whole construction is as rough as possible: Large nails; hinges with rough edges; no attempt to conceal, as would have been easy, the large lever on the dorsum of the hand.

The thumb is solid with the metacarpal part and rigid with flexion in a curve, evidently designed for holding a cylindrical object. The fingers, which show the normal relations of length, are firmly soldered to a single metal cylinder that works round a transverse pivot, which is continuous with the metacarpal portion. The fingers, like the thumb, have no interphalangeal joints, but are curved in flexion. Their palmar aspect, as is that of the thumb, is flat, evidently with the object of eliminating curves that would have given a less firm grip on an object. The fingers can be passively flexed till the pulp of the middle one reaches to within 3 cm. of the palm. The flexion can be graded in six stages, thanks to a ratchet placed round the cylinder from which the fingers spring. Into the teeth of the ratchet fits the free end of a metal arm, which articulates with a lever fixed to the dorsal trough of the wrist.

When the fingers are closed into a fist, pressure on a button at the inner margin of the hand, acting through a lever, frees the ratchet from the metal arm. Then there occurs immediate full extension of the fingers, probably due to a spiral spring enclosed in the cylinder to which the fingers are soldered, a spring which must be fixed to the transverse pivot round which the cylinder turns. This seems the logical explanation, for the actual mechanism, being enclosed in the cylinder, is invisible.

Extension, as well as flexion, can be adjusted in six grades and in each the movement is irreversible. Closure of the fingers leaves a quadrangular space between them and the thumb, but in the final stages it becomes triangular. This gives the optimum grip on a cylindrical object.

The hand was attached to the forearm by four metal plates, three of which, cracked at their bases, still remain attached to the wrist.

(To be concluded)

REFERENCES

[1] "Why do you offer me the left hand? Am I not worthy of knightly courtesy?"

[2] "Were you the emperor himself, you must be content with this. My right, though not useless in war, is insensible to the pressure of love; it is a part of my glove; you see, it is iron." *Deutsche Nat. Litt.*, Vol. 89; *Goethes Werke*, Berl., 8: 252, 1885.

[3] "Has rendered more service in the fight than ever did the original of flesh."

[4] Amputation in the days of Hippocrates is described by the following:

"When gangrene supervenes in a fracture, the soft parts separate quickly; as for the bones, they become detached at the limit of their exposure, but much more slowly. It is necessary to remove whatever dies first below the lesion from the the healthy parts, avoiding as far as possible pain, for patients die from fat embolus." Hippocrates, Mochlico, T. Oeuvres Complètes. Translated by Littré. Paris, 1844, 7: 379.

[5] Rivière, E. Prothèse chirurgicale chez les anciens. *Gazette des Hopitaux*, No. 132, Nov. 17, 1883.

[6] Jo. Thomae Minadoi Rhodigini. De Humani Corporis Turpitudinibus cognoscendis et curandis. Patavii, apud Franciscum Bolzettam, MDC, Lib. III, Cap. XIV, fol. 95 ret.:

Excogitarunt homines maioribus forte machinamentis deformitatis remedia, dum ferreas manus, in carnem naturalisque praecisae detruncataeque locum subdunt. Ego sane novi nobilem quendam virum, qui cum illi nescio quo casu ex insidiis utraque manus fuerit detruncata, ita ut sine manibus jam viveret, ferreas manus a quodam artifice obtinuit, tam apte cohaerentes, tanta arte et tot librationibus et nescibus conciliatas, ut cum illis pileum et capite amovere et rursum capite remittere et bursam solvere atque ligare et calamo nomen proprium perscribere posset. Alium quoque novi cui dextra manus bellici tubuli fulmine icta amputataque fuerit, qui ferream pariter manum sibi magna cum industria comparaverat, ita ut cum illa multa perageret, quae carnea olim manu etiam perficiebat.

[7] The Museo Stibbert is a museum of antiquities, collected by Mr. Stibbert, an Englishman, who long resided in Italy and at his death left all his possessions to the English Government, with the proviso that if they were not accepted, they should be given to the city of Florence. The English Government having refused the bequest, the city of Florence took possession of the Museum, which excels many in Italy and competes with the most famous abroad by the richness of its collection of weapons of every age and country. The magnificent catalogue of the Museum is the work of Alfredo Lensi, head of the Ufficio Antichita e Belle Arti of the city.

[8] The limbs are enumerated in the catalogue of the Museum as Nos. 3816, 3817, 3818, 3819, 3820 and depicted in Table 174.

[9] The number corresponds to that in the catalogue of the Museo Stibbert. The date is that thought probable by the Director of the Museum.

[10] "Only rare examples are found from the beginning of the sixteenth century. They were in frequent use in the second half and also for most of the seventeenth century." Angelucci—Catalogue of the Royal Armory at Turin, lvi, 1890, p. 69.

BOOK REVIEWS

No reviewer's testimony can add to the stature of Hugh Hampton Young, urologist. This work[1] emphasizes once more the everlasting indebtedness of the medical profession in general, and of practitioners of urology in particular, to the genius of the director of the Brady Urological Institute. To Young's sound practical judgment, begotten by an extraordinary experience, has in this instance been added that of Charles A. Waters, Assistant Roentgenologist in the same institute. The result is a work distinguished by acute analysis and scholarly presentation of what heretofore has been a relatively unexplored field of urological activity.

Much of the advancement of urological roentgenology originated at the Brady Urological Institute. The substitution of a harmless opaque medium for the highly toxic media first used in pyelography was the work of one of its graduates. There, too, was designed and constructed the first combined cystoscopic-roentgen-ray table, making the pyelogram a more simple and scientifically accurate process. Young's adaptation of the Bucky diaphragm, with grids running transversely instead of longitudinally, permitted the taking of stereoscopic films of the entire genitourinary system, and widely extended the diagnostic possibilities of urological roentgenology.

The scope of this work has purposely been narrowed, for two years ago the senior author published his monumental "Practice of Urology." The present volume treats specifically of urology in all its relations to roentgenology, and trenchantly discusses, *inter alia*, stereoscopy, ureterography, cystography, diverticulography, vesiculography, ampullography, vasography, topography of the kidneys, anatomy of the pelvis and calices, renal anomalies, renal tumors, traumatisms and foreign bodies of the urinary tract.

The opening chapter deals with the practical aspects of a combined roentgenological and urological study of the genitourinary tract. The technique of pyelography is lucidly set forth. Preference is given to the gravity method of introducing the opaque medium. The authors make a bilateral pyelogram unless the condition of the patient contraindicates it. They also procure stereoscopic views whenever possible, stressing their importance especially in renal tuberculosis.

The technique of cystoscopic examination is complete in its delineation. Respecting pyeloscopy, the reviewer believes that most urologists will agree with the authors in their estimate of the advantages and disadvantages of fluoroscopy in diagnosis; for, while it is interesting to observe the filling of the calices and visually to determine the emptying time, the actual opportunities for observing pathological conditions are few, the chance of error is not negligible, and no permanent record is obtained, unless the examination is prolonged to make pyelograms with consequent fatigue and danger to the patient. What is said in respect of the proper position of the patient upon the table will be appreciated by every urologist who has cystoscoped a patient upon the ordinary roentgen-ray table, since it is essential to make the pyelogram in the horizontal position as well as the vertical.

The section on renal fluoroscopy by Braasch, from an as yet unpublished monograph, is a convincing refutation of the objections often made against the use of fluorosopy in the removal of multiple renal stones. There is urged the value of both fluoroscopy and roentgenograms at the operating table, particularly in the case of calculus, and Quimby's method of using roentgen-ray films is explained.

[1] Urological Roentgenology. By Hugh H. Young M.D., F.A.C.S. and Charles A. Waters, M.D., Annals of Roentgenology, Vol. VII. Edited by James T. Case. 4 to, cloth. Pp. 545, 494 illus. N. Y., Paul B. Hoeber, Inc., 1928.

The authors remind us of many anatomical and physiological aspects of urological problems, the significance of which may be overlooked because of the proclivity of the specialist to exaggerate factors peculiar to the subject of his intensive study. Thus, in the interpretation of ureterograms, it is remarked that peristalsis often continues during the taking of the film, and waves of contraction will be seen in films of a normal ureter, creating at times the false impression of the presence of stricture. Again, it is noted that, during exposure time, the movement of a ureter filled with opaque medium will occasionally cast a wider shadow than the normal, giving the erroneous appearance of ureteral dilatation.

The discussion of urogenital infections and infestations, of tuberculosis of the genitourinary tract, and of urinary lithiasis is vivid and concrete. Here are none of the generalities wherein error lurks. The case method is the vehicle of development of these topics and the cases are made luminous by striking illustrations. Here as elsewhere the illustrator, Mr. William P. Didusch, has done his part admirably. From the colored plate of hypernephroma, with hydronephrosis and diffuse glomerular nephritis, which forms the frontispiece, to the deliniation of the pyelogram which it accompanies, shown on page 420, the illustrations are characterized by a fidelity to detail and a pictorial sense that visualize the minutest anatomical aspects of the subject.

The publishers have given the volume the sumptuous dress to which a work of such merit is entitled.

It is not too much to say that no practitioner of urology will want to be without this work.

BOOKS RECEIVED

All books received by The American Journal of Surgery are listed in this column as soon as possible after their receipt and this must be considered as adequate acknowledgment. Books that the Editor considers of special interest to our readers will be reviewed in a later issue.

Annual Report of the Surgeon General of the Public Health Service of the United States, for the Fiscal Year 1928. Crown 8vo, cloth. Pp. 352. Washington, D. C., Treasury Dept., 1928.

Diabetic Surgery. By Leland S. McKittrick, M.D., F.A.C.S., and Howard F. Root, M.D. 8vo, cloth. Pp. 279, 81 illus. Phila., Lea & Febiger, 1928.

Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus. By William Harvey, M.D. Tercentennial Edition. (English translation and annotations.) By Chauncey D. Leake. 8vo, cloth, Pp. 168, 10 illus. Springfield, Ill., Charles C. Thomas, 1928.

Handbook of Microscopical Technique. Edited by C. E. McClung, PH.D. 8vo, cloth. Pp. 510, 43 illus. N. Y., P. B. Hoeber, Inc., 1929.

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THIS MONTH'S CONTRIBUTORS

BERENS, CONRAD, M.D., F.A.C.S., N. Y.
Chief Surgeon, New York Eye and Ear Infirmary; Consulting Ophthalmologist, Neurological Division, Vanderbilt Clinic; Director, Ophthalmology, Midtown and Pan American Hospitals; Consulting Ophthalmologist, U. S. Veterans and New York Infirmary for Women and Children.

BOOTHBY, WALTER M., M.D., ROCHESTER, MINN.
Associate Professor of Medicine, the Mayo Foundation; Head of the Section on Metabolism. Author, with Irene Sandiford, of: "Basal Metabolic Rate Determinations."

BREITNER, D. B., M.D., VIENNA
Von Eiselberg Clinic.

CARSON, W. J., M.D., MILWAUKEE
Instructor in Surgery, Marquette University; Member, Surgical Staff, Marquette University Hospital; St. Luke's Hospital.

CARTER, WILLIAM WESLEY, M.D., F.A.C.S., N. Y.
Visiting Laryngologist and Otologist, Bellevue and Allied Hospitals (Gouverneur Hospital); Consulting Laryngologist & Otologist, South Side Hospital; Author of numerous monographs on Deformities of the Nose and Bone Transplantation.

CHAFFIN, RAFE C., M.D., F.A.C.S., LOS ANGELES
Instructor and Assistant Professor in Gynecology, College of Medical Evangelists; Attending Gynecologist, Los Angeles General Hospital.

CLUTE, HOWARD M., M.D., F.A.C.S., BOSTON
Surgeon, Lahey Clinic; Assistant Surgeon, New England Deaconess Hospital; Assistant Surgeon, New England Baptist Hospital.

COLE, WARREN H., M.D., ST. LOUIS
Assistant Professor of Surgery, Washington University School of Medicine; Assistant Surgeon, Barnes, St. Louis Children's and St. Louis Maternity Hospitals; Visiting Surgeon, St. Louis City Hospital.

DECOURCY, JOSEPH L., M.D., CINCINNATI
Attending Surgeon, Good Samaritan Hospital.

DICKINSON, ARTHUR M., M.D., F.A.C.S., ALBANY, N. Y.
Junior attending Surgeon, Memorial Hospital.

ELSE, EARL J., PH.G., M.S., M.D., PORTLAND, ORE.
Chairman, Department of General Surgery, Clinical Professor of Surgery, University of Oregon Medical School.

GAGE, I. M., M.D., NEW ORLEANS
Assistant Professor of Surgery, Tulane University Medical School.

GRAY, S. H., M.D., ST. LOUIS
Assistant Professor of Pathology, Washington University School of Medicine; Assistant Pathologist to Barnes and St. Louis Childrens Hospitals.

HAINES, SAMUEL F., M.D., ROCHESTER, MINN.
Instructor in Medicine, The Mayo Foundation; First assistant in one of the Sections of Medicine, The Mayo Clinic.

HUTTON, JAMES H., M.D., CHICAGO
Chief of Medical Service, Woodlawn Hospital.

KEMPER, CONSTANTINE F., M.D., DENVER
Instructor in Medicine, University of Colorado School of Medicine; Member of Hospital Staff; University of Colorado, Denver General, St. Luke's, St. Joseph's, Childrens and Presbyterian Hospitals. Editor, *Colorado Medicine*.

KING, B. T., M.D., SEATTLE, WASHINGTON

KLING, DAVID H., M.D., LOS ANGELES
Director of Laboratories, Golden State Hospital; Visiting Physician to the Jewish Consumptive Sanitarium, Duarte, Calif.

LOSEY, RAY B., M.D., PH.G., N. Y.
Senior Assistant Surgeon, New York Eye and Ear Infirmary; Associate Director of Ophthalmology, Pan-American Hospital; Ophthalmic Surgeon, Midtown Hospital.

MORTIMER, EGBERT L., M.D., BALTIMORE
Associate Surgeon, St. Agnes Hospital.

ORMOND, JOHN K., M.D., F.A.C.S., DETROIT
Surgeon in Charge of Division of Urology, Henry Ford Hospital.

PUTTI, V., M.D., BOLOGNA
Professor of Orthopedic Surgery, University of Bologna; Director of the Rizzoli Institute.

STEVENS, A. E., M.D., N. Y.
Associate Professor of Urology, Cornell University; Attending Surgeon, Director of Department of Urology, Bellevue Hospital; Consulting Urologist, Beekman Street, Reconstruction and Nursery & Childs Hospitals; also Stamford and Tarrytown Hospitals; and Somerset Hospital (Somerville, N. J.).

VALENTINE, J. J., M.D., N. Y.
Professor of Urology, New York Polyclinic Medical School & Hospital; Assistant Adjunct Visiting Surgeon, Department of Urology, Bellevue Hospital; Urologist, Bronx General Hospital; Director of Urology, Pan-American Hospital.

WOLFSON, WILLIAM J., M.D., F.A.C.S., BROOKLYN
Associate attending Surgeon, Jewish and United Israel Zion Hospitals.

WOMACK, NATHAN A., M.D., ST. LOUIS
Assistant in Surgery, Washington University School of Medicine; Resident Surgeon, Barnes Hospital.

WOODRUFF, STANLEY R., M.D., N. Y.
Associate Professor of Urology, N. Y. Post-Graduate Medical School and Hospital; Attending Urologist to Christ Hospital, Jersey City and Bayonne Hospital, Bayonne, N. J.; Consulting Urologist to St. Mary's, Hoboken and Greenville Hospitals, Jersey City.